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Inside This Issue

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Since Invest in ME was first formed, and especially since our successful 2007 biomedical research conference, we have felt that international cooperation is the likely key to providing the significant progress required for people with ME. It is by uniting across countries and continents that we can better tackle the problems in healthcare services regarding lack of up-to-date information on ME/CFS which are apparent across Europe. Increased knowledge of the findings from the latest biomedical research needs to filter down to doctors, nurses and other healthcare staff who are at the front end of examining and treating people with ME. Eventually that information will work its way into establishment organisations and into government policy – perennially the last areas which accept the latest evidence.

International cooperation is also necessary between researchers and research establishments as this makes better use of scarce resources (funding, research units etc.) to achieve the necessary strategic approach to research into ME/CFS.

Since the last Invest in ME international biomedical research conference in London in May Invest in ME has been working with our European colleagues and now the European ME Alliance has been set up. This new collaboration is an effort to campaign for funding for biomedical research into ME but it is also going to provide a one- stop site for correct and up-to-date information on ME for all Europeans. We look forward to increasing the benefits for people with ME and their families via our efforts to cooperate across national boundaries.

Invest in ME now announces our fourth International ME/CFS Conference for May 2009. We hope to build on the success of the conferences of the past years including the 2008 conference dealing with Sub Groups of ME/CFS – surely the way forward for biomedical research.

The severely affected people with ME are neglected by healthcare organisations and by the establishment authorities responsible for funding research. Many believe it is only by examining severely affected patients that a cure will be found for this illness yet those establishment organisations responsible

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for medical research into ME/CFS continually allow flawed research which purposely ignores the severely affected in their selection criteria. Our own support work with severely affected people with ME in the last 9 months proves that a section of patients receive no attention from the healthcare system – and this is in London.

Our International ME/CFS Conference in May will try to focus more on those severely and moderately affected patients in the hope of attracting attention to the need for research into severe ME.

In the Journal Greg Crowhurst has provided a nursing model for severe ME and Sue Pearkes has contributed an article to increase understanding of the issues faced by wheelchair users – a very interesting and different perspective which ought to be read by those who are responsible for providing management strategies for people with ME.

The lack of proper attention given to severely affected people with ME is highlighted by our new book project – Lost Voices - a book developed over the last year and using the power of pictures to supplement the moving stories of people with ME who have been left on the medical scrapheap. Lost Voices encapsulates the tragedy of this illness and the way in which people with ME are left to deal with this illness by themselves with no hope of a future.

We welcome in this Journal articles from distinguished experts on ME/CFS who have presented at our international ME/CFS conferences.

Dr Leonard Jason has kindly submitted a paper examining differences between blood and non-blood relatives in five illnesses, including ME/CFS. The findings show genetic and environmental factors are associated with ME/CFS. Research into diagnosing and treating ME/CFS needs to ensure that proper sub groups are being used.

In his Letter from America Dr Martin Lerner addresses concerns among ME/CFS physicians endeavouring to help patients.

Dr Bruce Carruthers has provided a very thought provoking and thorough insight into the way researchers and clinicians should work.

And so to NICE. If anyone had any doubts about the inappropriateness of the NICE Guidelines for ME/CFS then we have articles in the Journal which simply show why NICE have again failed the people they are meant to serve and why their guidelines are plainly unfit.

We have translated an article from the Norwegian ME Association which clearly shows the failings in the NICE guidelines. It is a sad fact that some European healthcare services are under the impression that adoption of the UK NICE Guidelines for ME/CFS would be a sensible approach and could save money. This impression is false and will lead to neither appropriate nor economical services being supplied.

NICE ignored the data which existed regarding ME/CFS in favour of a one-size fits all package of rehashed psychiatric paradigms – the same paradigms which have been promoted by the government, MRC and psychiatrists for years and which have not only done damage to people with ME but also completely failed in their supposed intent.

Patients bringing legal actions - familiar territory for NICE - an indication of an organisation which is out of step with patients' needs. NICE needs to ensure it stays current in its knowledge regarding ME, just as front-line doctors and other healthcare staff need the same currency to perform adequately any work related to people with ME. Hopefully the Journal of IiME will help in some small way to keep this currency in tact. We hope the New Year will begin with the NICE guidelines for ME being consigned to the shredder – a fate, unfortunately, which they thoroughly deserve.

Invest in ME look forward to the New Year where we have a new book, a new European organisation campaigning for people with ME and a new international ME/CFS conference.

All at IiME wish our readers a Happy Christmas and a cure for the New Year.

ME/CFS and Family Medical History
Family Illnesses Among People with ME/CFS: Blood Versus Non-Blood Relatives

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ABSTRACT

Most research examining the family history of persons with ME/CFS have primarily investigated differences between individuals with ME/CFS and control groups without the illness. Research examining differences between blood and non-blood relatives might contribute to understanding genetic and environmental etiologic factors. The current study investigated the occurrence of five illnesses (i.e., diabetes, Lupus, Multiple Sclerosis, Fibromyalgia and ME/CFS) among blood and non-blood relatives of individuals with ME/CFS. Family history of medical illness was obtained from self report data completed by participants. We determined the number of participants reporting a family history of diabetes, Lupus, Fibromyalgia, Multiple Sclerosis, and ME/CFS between the blood-related family members and non-blood-related family members of participants with ME/CFS. There was a greater prevalence of diabetes, Lupus, Fibromyalgia and ME/CFS among blood relatives than non-blood relatives. The findings of this study suggest that both genetic and environmental factors are associated with ME/CFS.

Keywords:

Family Histories, Autoimmune; endocrine; ME/CFS.

Professor Leonard Jason

Professor of Clin. & Community Psychology, Director, Center for Community Research, DePaul University, Chicago

Dr. Leonard Jason, Ph.D., is among the most prolific of all CFIDS researchers. For more than a decade, Dr. Jason and his team at DePaul University's Centre for Community Research have worked to define the scope and impact of CFS/ME worldwide.

Professor Jason presented at the IiME International ME/CFS Conference 2008 in London.

Research on the etiology of ME/CFS (Myalgic Encephalomyelitis/Chronic Fatigue Syndrome) suggests that endocrinological factors may influence the development of this illness (Friedberg & Jason, 1998). Endocrine abnormalities such as thyroid dysfunctions and low functioning of the hypothalamic-pituitary-adrenal axis have been linked to the etiology of ME/CFS (Addington, 2000; Demitrack et al., 1991). Other studies have found associations between ME/CFS and

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immune system low-level activation, abnormalities in T-cells, reduced natural killer cells activities and IgG1/IgG3 deficiencies (Bates et al., 1995; Buchwald et al., 1992; Jason, Torres-Harding et al., 2007; Patarca-Montero et al., 2000). Findings in neurological studies have suggested that impaired autonomic nervous system functioning may impact the development of ME/CFS (Freeman & Komaroff, 1997; Pagani & Lucini, 1999). Unfortunately, there is a lack of consistency in findings from different studies (Torres-Harding et al., 2005).

Family studies of persons with ME/CFS have examined endocrinological, immunological, and neurological associations with ME/CFS. Endicott (1999) assessed the family histories of 45 psychiatric patients diagnosed with ME/CFS in comparison to 90 psychiatric patients without the condition and 45 randomly chosen patients. The results indicated a higher prevalence of cancer, autoimmune disorders, and ME/CFS related conditions among parents of those with ME/CFS and no differences in psychiatric disorder history (Endicott, 1999). In another family history study of persons with ME/CFS, Walsh, Zainal, Middleton, and Paykel (2001) compared 25 persons with ME/CFS to a matched control group of 36 participants who had inflammatory bowel disease, Crohn's disease or ulcerative colitis. The findings indicated that persons with ME/CFS were more likely to have a family history of chronic fatigue and ME/CFS than the control group.

Endocrine system dysregulations have also been noted in family history of persons with ME/CFS. Torres-Harding, Jason and Turkoglu (2005) examined family medical histories of people with ME/CFS and found that 50 percent of people with ME/CFS had a relative with an endocrine/metabolic illness compared to only 28 percent of the non-ME/CFS group. The illnesses indicated were diabetes/diabetes mellitus, thyroid-related conditions and grave's disease (Torres-Harding et al., 2005), with diabetes/diabetes mellitus being the most frequently reported illness. As an endocrine/metabolic disorder, diabetes interferes with the body's process of digesting food for both growth and energy. The most recent statistics indicate that 8 percent of the U.S. population have been diagnosed with diabetes (American Diabetes Association, 2008).

Examining the family history of individuals with ME/CFS may assist in determining the etiology or risk factors associated with ME/CFS. A combination of genetic and environmental factors may be associated with ME/CFS. A study of 124 monozygotic and dizygotic twins suggested that both genetic and environmental components influence the onset of fatigue (Hickie et al., 1999). A particular genetic component was found to predict fatigue and increased immune responsiveness, whereas an environmental component predicted fatigue and decreased immune responsiveness.

Most family history studies reviewed above have compared individuals with ME/CFS to a control group without ME/CFS. The current study investigated the family history among blood and non-blood relatives. The occurrence of diabetes, Lupus, Multiple Sclerosis, Fibromyalgia and ME/CFS among blood and non-blood relatives of persons with ME/CFS was examined. It was hypothesizedthat family history of these illnesses would be higher in blood relatives than non-blood relative.

Method

Participant Recruitment.

Study participants were derived from a larger treatment trial investigating the effectiveness of non-pharmacologic interventions for individuals with ME/CFS (Jason et al., 2007). Participants were recruited from a variety of sources, including physician referrals. Information about the non-pharmacologic treatment trial study was disseminated to medical colleagues through mailings and phone communication. In addition, study announcements for new participants were placed in local newspapers and recruitment offers were made at local

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ME/CFS support group meetings. These efforts were continued throughout the study period until the target enrollment numbers were achieved. One hundred and fourteen individuals were recruited. Of the 114 individuals, 46% were referred by physicians, 34% were recruited by media (newspapers, TV, radio, etc.), and 20% stemmed from other sources (e.g., heard about the study from a friend, family member, person in the study, etc.). Twenty-four additional individuals who were screened were excluded due to a variety of reasons (i.e., lifelong fatigue, less than 4 Fukuda symptoms, BMI > 45, melancholic depression or bipolar depression, alcohol or substance abuse disorder, autoimmune thyroiditis, cancer, lupus, rheumatoid arthritis).

Initial Screening. All participants were required to be at least 18 years of age, not pregnant, able to read and speak English, and considered to be physically capable of attending the scheduled sessions. Bedridden and wheelchair bound patients were excluded due to the practical difficulties of making appointments. Referrals to local physicians who treat ME/CFS and to support groups were offered to these individuals. After a consent form was filled out, prospective participants were initially screened by the third author, using a structured questionnaire. Because ME/CFS is a diagnosis of exclusion, prospective participants were screened for identifiable psychiatric and medical conditions that may explain ME/CFS-like symptoms. These measures were completed at DePaul University and took approximately two hours. After the initial interview was completed, the patients' information was reviewed to ensure that they met all eligibility requirements.

If found to be eligible for the study, all participants attended a medical appointment with the study physician in order to confirm the diagnosis of ME/CFS. After confirmation that the individual fully met the criteria for ME/CFS according to the Fukuda et al. (1994) case definition, individuals completed a battery of baseline measures (described below). They were also assigned randomly to one of four treatment conditions, and completed measures at three follow-up testing periods. However, only the data obtained at baseline was considered in the current investigation.

Measures

The ME/CFS Questionnaire.

This screening scale was initially validated by Jason et al. (2007). Hawk, Jason, and Torres-Harding (2007) recently revised this ME/CFS Questionnaire, and administered the questionnaire to three groups (those with ME/CFS, Major Depressive Disorder, and healthy controls). The revised instrument, which was used in the present study, evidences good test-retest reliability and has good sensitivity and specificity (Hawk et al., 2007). This scale was used to collect demographic, health status, medication usage, and symptom data, and it used thedefinitional symptoms of ME/CFS (Fukuda et al., 1994). For each Fukuda et al. (1994) case definition symptom, rate the intensity of each symptom they endorsed on a scale of 0 to 100, where 0 = no problem and 100 = the worst problem possible. The mode of illness onset was derived from an item on this measure. Illness onset duration of one month defined the sudden illness onset group while onset duration of longer than one month signified gradual illness onset.

Medical Examination: The physician screening evaluation included a general and neurological physical examination. Laboratory tests in the battery were the minimum necessary to rule out other illnesses (Fukuda et al., 1994). Laboratory tests included a chemistry screen (which assesses liver, renal, and thyroid functioning), complete blood count with differential and platelet count, erythrocyte sedimentation rate, arthritic profile (which includes

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rheumatoid factor and antinuclear antibody), hepatitis B, Lyme disease screen, HIV screen and urinalysis. A tuberculin skin test was also performed. If the TB skin test was positive, a follow-up chest x-ray was conducted to rule out tuberculosis. The project physician performed a detailed medical examination to detect evidence of diffuse adenopathy, hepatosplenomegaly, synovitis, neuropathy, myopathy, cardiac or pulmonary dysfunction. This medical examination was used to confirm the diagnosis of ME/CFS, according to the Fukuda et al. (1994) criteria and to rule out exclusionary medical conditions.

Family history of illness: Family history of medical illness was obtained from self report data completed by participants. Participants were asked: "have any of your relatives been diagnosed with the following medical conditions?" Medical conditions included diabetes, Lupus, Multiple Sclerosis, Fibromyalgia, and ME/CFS. The participants were asked to report on these conditions for both blood (i.e., biological mother, father, grandparents, sibling, children, other) and non-blood relatives (i.e., spouse, step-parent/primary care giver, adopted children and other). Seventeen possible blood relatives include: mother, father, daughter, son, brother, sister, aunt, uncle, grand father, grand mother, great grand father, great grand mother, great aunt, great uncle, nephew, niece, and cousin. Seventeen possible non-blood relatives include: **s**pouse, mother in-law, father in-law, adoptive mother, adoptive father, adopted son, adopted daughter, step-mother, step-father, step-daughter, step-son, step-brother, step-sister, sister in-law, brother in-law, grand father in-law, and grand mother in-law. We computed the number of participants reporting a family history of each illness.

Statistical Analyses

The occurrence of each medical illness (i.e., diabetes, lupus, Fibromyalgia, Multiple Sclerosis, and CFS) between the blood-related family members and non-blood-related family members of these participants with ME/CFS was examined with McNemar tests. The effect size was computed using a procedure described by Green and Salkind (2003) for the McNemar Test. The difference in the proportions of participants who fell into the two family relative types was computed for each illness to obtain the effect size index. Cohen's (1988) guidelines for interpreting effect size; 0.01 = small effect, 0.06 = moderate effect and 0.14 = large effect, was used to estimate the strength of the effect sizes.

Results

The McNemar analyses indicated significant higher percentages of diabetes, Lupus, Fibromyalgia and ME/CFS among blood than non-blood relatives (see Table 1). Of the total of 114 participants, 42.1% (N = 48) reported that they had blood-related family members who had diabetes, whereas 4.4% (N = 5) reported having non-blood family members with diabetes ($p < .01$ with an effect size index of 0.38). A person could have more than one family member of non-family member with diabetes, and for the blood relatives, there were a total of 75 cases of diabetes, whereas there were a total of only 5 cases for non-blood relatives. Among the 48 individuals with ME/CFS who had a blood relative with diabetes, 15 (31.3%) indicated that they had 2 or more blood relatives with diabetes (none of the non-blood relatives had 2 or more relatives). Most cases of diabetes occurred for parents (especially the father), with fewer cases among siblings, and with only one report of a child with diabetes. Among the 114 participants who had diagnosed ME/CFS, one reported having diabetes and two reported having borderline diabetes. The individual with diabetes had a mother with diabetes, but the two participants who reported borderline

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Table 1

Family History of Medical Illness among Blood vs. Non-blood relatives of Persons with ME/CFS

History	Blood Relative N (%)	Non-blood Relative N (%)	Significance
Diabetes	48 (42.1%)	5 (4.4%)	**
Lupus	8 (7.0%)	1 (0.9%)	*
MS	5 (4.4%)	1 (0.9%)	
Fibromyalgia	17 (14.9%)	3 (2.6%)	**
CFS	6 (5.3%)	0 (0.0%)	*

** indicates significant at .00 level

* indicates significant at .05 level

diabetes did not have familial history of diabetes.

Examining the occurrence of Lupus, 7.0% (N = 8) of the participants indicated having blood-related family members who have Lupus as compared to .9% (N =1) for non-blood family members ($p < .05$, an effect size index of .06). Regarding Fibromyalgia, 14.9% (N = 17) of the participants indicated that they have blood relatives with this illness whereas 2.6% (N = 3) reported having a non-blood relative with Fibromyalgia. ($p < .01$, with an effect size index of .12). Approximately, 5.3% (N = 6) of the participants reported having blood relatives with ME/CFS whereas none were indicated for non-blood relatives ($p < .05$ with an effect size index of .05). For Multiple Sclerosis, no significant differences occurred between those with blood relatives (4.4%, N = 5) and those with non-blood relative (.9%, N = 1).

Discussion

A higher percentage of diabetes, Lupus, Fibromyalgia and ME/CFS were reported among blood relatives than non-blood relatives of people with ME/CFS. The largest difference was found for diabetes, suggesting that a familial predisposition to endocrine system impairment may contribute to the development of ME/CFS. Similar findings emerged elsewhere (Torres-Harding et al., 2005), and these studies might represent the influence of both genetic and environmental factors. We did not find a high percentage of the participants with ME/CFS to have diabetes, as only one participant had diabetes and two reported borderline diabetes. Certainly, it is important to follow-up these individuals to determine whether more people with ME/CFS develop diabetes over time.

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Endicott (1999) reported a higher rate of autoimmune disorders in parents of persons with ME/CFS whereas Torres-Harding et al. (2005) did not find any differences in familial autoimmune vulnerabilities among persons with ME/CFS and a control group. In the current study, we examined two autoimmune diseases: Lupus and Multiple Sclerosis. Whereas a significant difference was found for Lupus, there were no significant differences in the familial history of Multiple Sclerosis between blood relatives and non-blood relatives. Low power and small sample sizes might have been the reasons for not being able to detect statistical differences for Multiple Sclerosis. Certainly, there is a need for larger samples to determine if these findings are replicated by other investigators.

Both Endicott (1999) and Walsh et al. (2001) found that persons with ME/CFS were more likely to report chronic fatigue-like illnesses than control groups. In contrast, Torres-Harding et al. (2005) found no significant differences in family background for these illnesses. In the present study, there were more familial reports of ME/CFS for blood relatives than non-blood relatives indicating interesting familial links predisposing individuals toward the development of ME/CFS. Many studies have documented that Fibromyalgia tend to co-occur with ME/CFS (Brown & Jason, 2007; Jason et al., 2000; Jason et al., 2001) but little is known about familial history of Fibromyalgia. The current study found significantly higher rates of familial Fibromyalgia history among blood relatives than non-blood relatives suggesting other predisposing factors in the development of ME/CFS.

The current study was limited by several factors, including the assessment of only five familial illness histories. It is possible that there may be other illnesses that were not assessed in this study. In addition, recall bias tends to impact the self report data, and it is certainly possibly that individuals tend to recall illnesses of blood relatives more than non-blood relatives' illnesses. In addition, this study did not include reports of the demographic information of the relatives, which could have helped to examine other possible sociodemographic factors. The lack of a matched control group by age and race is another limitation of this study. The results may have been impacted by the lack of equal number of blood and non-blood relatives. It is unclear whether people with ME/CFS have more blood or non-blood relatives, so it is at least possible that the results were influenced by this finding.

The most serious potential confound in this study was that it could be argued that there are more blood relatives than non-blood relatives. Yet the findings, particularly for diabetes, would even take this into account. If 8% of the population has diabetes (American Diabetes Association, 2008), than among the 114 people in the sample with ME/CFS, there would be 228 parents, and about 18 expected cases of diabetes among these 228 parents. However, among the fathers and mothers of the sample, there were 34 cases of diabetes (and all of these cases came from blood relatives), suggesting a rate more than double what would have been expected, which would have been 18. In addition, if one were to take all cases of non-blood relatives, there were only 5 cases of diabetes. In contrast, there were 75 cases for those with blood relatives. This difference is large, but one might still question whether there were more biological relatives than non-biological relatives. This concern could also be addressed if one were to limit the number of biological relatives for each person with ME/CFS. For example, if one were to just focus on one type of blood relative, the father of the person with ME/CFS, and compare the 114 fathers of the people with ME/CFS to all the non-blood relatives of the 114 people with ME/CFS, there certainly would be more people in the non-blood group than the blood group. Even though in this comparison there were more non-blood relatives, we only

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found 5 total cases of diabetes for all non-blood relatives, whereas for the fathers of people with ME/CFS, there were 20 cases. These findings suggest that at least for diabetes, the outcomes are not likely due to there being more blood relatives than non-blood relatives.

Furthermore, one could argue that a person may have more non-blood relatives than blood relatives. According to the United States Census Bureau (2000), the average family size in the United States is 3.14. Using this statistic, after four generations of two parents having one child, a fourth generation person would have 14 blood relatives. These 14 blood relatives are the person's: 8 great grandparents, 4 grandparents, mother and father. However, when this individual marries their spouse, assuming their spouse is also a fourth generation person from one child families, this individual will gain 15 non-blood relatives. These 15 non-blood relatives include their spouse, and their spouse's 8 great grandparents, 4 grandparents, and 2 parents. Therefore, it is at least conceivable that there might be as many non-blood relatives, if not more, than blood relatives.

In general, the findings of this study found that family members who are related by blood have several medical illnesses at higher rates than those who are non-blood related. Certainly, the findings are strongest for diabetes, and it is always possible that recall bias influenced the results. However, the robust nature of the outcomes indicates this is an area worthy of future investigations, and having medical work-ups of both blood and non-blood relatives would strengthen research. There are policy implications of this work, for if individuals with ME/CFS do have blood relatives with more medical illnesses, it is possible that both genetic and environmental factors need to be considered when understanding the etiology of this illness and when providing treatment for those with this illness.

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Letter from America

A Letter to My English Friends:

By Dr. A. Martin Lerner

Thank you for your kindnesses to me during my visit to London to participate in your May 2008 International ME Conference. I had the unique opportunity to meet and speak personally with many of you.

Thank you again.

I address concerns among CFS physicians endeavoring to help our CFS patients. We all agree with criteria for the internationally accepted CFS definition.

Today, the "gold" standard for evidence-based medicine proof of cause depends upon a trial of treatment with two similar equal number groups of patients, matched for age, time and place. One equal group receives the treatment option-in-question, and the second equal group receives a placebo. (We further know in CFS that the placebo improvement healing rate is 19%!) If the treatment group of the proposed randomized blinded trial improves in a much larger percentage, and, if this trial is repeated by a second independent group of investigator physicians, everyone would accept that the treatment in question was useful.

To date "useful" CFS treatments are psychotherapy and graded exercise. (I am omitting my own studies for now.) In Europe I believe that the tentative leading cause of CFS is "CFS is a psychiatric condition, a neurosis." Neither of these courses, graded exercise or psychotherapy with or without psychotropic medicines, leads to a normal life for the CFS patient.

Dr Martin Lerner

Dr Martin Lerner is Clinical Professor Wayne State University School of Medicine

Dr Lerner is certified by the American Board of Internal Medicine and is an Infectious Disease Specialist.

Dr. Lerner has published over 10 papers since 1993 on the role of subclinical myocarditis in a subset of CFS patients. He has also reported success with long courses of antiviral therapy in patients with chronic EBV and CMV infections..

Dr Lerner presented at the IiME International ME/CFS Conference in London in 2008.

- An evidence-based truth according to the famous polymath, David Hume requires cause, etiology, and this requires
- A) to be always followed by,
- B) a necessary condition.

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Letter from America

A Letter to My English Friends: (continued)

1) David Hume's theorem was met by the sputum culture isolation of the "tuberculosis" bacterium, and then, the transference of this organism to produce tuberculosis in an experimental animal.

2) Likewise, typical bacterial lobar pneumonia was cured by administration of penicillin to the sick patient with pneumonia.

The conclusions are:

1) the tubercle bacillus causes tuberculosis.

2) penicillin cures lobar pneumonia.

All patients with any illness, including CFS, are saddened because they, the CFS patients, in particular, are not well. This "illness-caused depression" is not unique to CFS disease. Likewise, exercise intolerance is universal in all CFS patients.

CFS patients have a genetic homogeneity (Jonathan Kerr's work, our London conference 2008): an immunologic cacophony: abnormal tilt table tests (neuro-humoral reflexes): increased RNase L lymphocyte activity: and many other abnormal biological findings consistent only with a non-psychologic cause. Sadness, depression, does not cause any of these physiologic abnormalities. There is no immunologic disarray, increased RNase L in blood or elsewhere, abnormal tilt table test or uniform genetic propensity in the array of psychiatric disease.

However, the sore throat, lymph node enlargement and tenderness, and overwhelming fatigue of CFS fit many of the criteria of the illness "infectious mononucleosis" which is caused by a first-episode experience with Epstein-Barr virus, usually in young persons. Another similar appearing mononucleosis-like illness is caused by cytomegalovirus. Each of these viruses cause a similar clinical appearance.

In May at your International Conference, I reviewed a published (now distant sentinel study (1997) of CFS patients with elevated serum IgG antibody titers) to cytomegalovirus infection whom I treated with intravenous ganciclovir (valcyte orally was not yet available). I reviewed with you in London two similar studies of CFS patients with similar elevated serum antibody to Epstein-Barr virus. I treated the Epstein-Barr virus CFS patients with valtrex and repeated the valtrex study with a blinded randomized placebo controlled trial. In these pilot studies, ganciclovir was strikingly effective in cytomegalovirus CFS patients, and valtrex was similarly effective in Epstein-Barr virus CFS patients. Earlier, I had published the hypothesis (1997) of specific Epstein-Barr, cytomegalovirus or Human Herpesvirus 6 etiology, in single or multiple infections for CFS. Montoya (Stanford University 2006) later also demonstrated that valcyte was beneficial to patients with Human Herpesvirus 6 CFS.

In May of 2008, I presented at your ME International CFS symposium 124 CFS patients cared-for at my CFS treatment center, 2001 – 2007. I looked for elevated serum evidence of all three viruses, Epstein-Barr virus; cytomegalovirus and Human Herpesvirus 6 in every patient. All CFS patients met International Criteria for CFS diagnosis. No known cause for their CFS illnesses could be found by all conventionally accepted methods. Some of these CFS patients had Epstein-Barr virus, but no cytomegalovirus or Human Herpesvirus 6, and conversely, for other CFS patients with cytomegalovirus infection or human herpesvirus 6 infection. The majority (approximately) 2/3 had

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Letter from America

A Letter to My English Friends: (continued)

evidence of several (of the three viruses) simultaneously. I treated my patients specifically by their evidence of the specific virus, and (and this had not been done before), I treated CFS patients, regardless of how long they had been ill, carefully, for at least twelve months. I carefully followed each patient to avoid possible toxicities of both valcyte and valtrex. There was no harm to any CFS patient with these cautions. Previously, Epstein-Barr alone had been considered to be the possible cause of CFS, and trials of treatment, were limited to ONE MONTH. The result of this "then" state-of-the-art evidence based trial was "no benefit, no Epstein-Barr virus cause for CFS." With our knowledge today, this early trial, published in the New England Journal of Medicine was misconceived. CFS is a 3 herpesvirus disease! Longer treatment than one month is needed.

Of my 124 CFS patients, the average duration of specific antiviral treatment was 2.9 years, and as presented to you in London, over seventy percent of my patients enjoyed sustained improvement, so that they no longer met international criteria for diagnosis of CFS. The validated metric for measuring the severity of CFS fatigue was the Energy Index Point Score (EIPS). For each average EIPS, at three month intervals, there were an average of 46 CFS patients for each of the 24 three month intervals of the 6 year study.

There is a 2:1000 chance of error in these data, or 998 chances of 1000 that CFS is caused by one or several of the three herpesviruses, Epstein-Barr virus, cytomegalovirus or Human Herpesvirus 6. It now is evident that we have the cause and treatment for CFS. This is evidence-based cause(s) for the complex Chronic Fatigue Syndrome disease.

There is also a Group B CFS disease. Perhaps, this subject can be the theme of a second "Letter to My English Friends," thank you again for inviting me to London, May 2008.

Sincerely yours,

A. Martin Lerner

With the invaluable help of the A. Martin Lerner CFS Foundation.

Facts About ME

In the UK, patients with autoimmune features and neurological signs and symptoms are usually the most sick and as such they are excluded from studies of "CFS" or chronic fatigue undertaken by psychiatrists, so the results of UK studies from which such patients are excluded are not representative of the true situation.

A particularly important piece of research in these patients has demonstrated sensitivity of the vascular endothelium to acetylcholine (a major neurotransmitter and vascular dilator) and this finding may have implications for many other cholinergic pathways (which are extensive throughout the body). (58)

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from

WHAT IS ME? WHAT IS CFS?

INFORMATION FOR CLINICIANS AND LAWYERS

Marshall, Williams and Hooper

http://www.investinme.org/Article-020%20What%20is%20ME%20What%20is%20CFS.htm

The European ME Alliance

The European ME Alliance is a collaboration of ME organisations within Europe who have the common aim of promoting biomedical research into Myalgic Encephalomyelitis (known as ME or ME/CFS) and increasing awareness of this debilitating neurological illness.

The European ME Alliance (EMEA) has the following objectives –

- To establish correct recognition of myalgic encephalomyelitis as an organic illness requiring biomedical research to treat and cure
- To establish correct diagnosis of patients
- To establish specialised biomedical centres for education/treatment/cures

Myalgic Encephalomyelitis is defined by the World Health Organisation as a neurological illness (code WHO-ICD-10-G93.3). The varying symptoms experienced by many severe ME sufferers may include: -

- post-exertional malaise and loss of muscle power with delayed and prolonged recovery
- general chronic weakness of limbs
- neurological disturbances
- cognitive problems such as memory loss & concentration difficulties
- problems with balance and fine motor control
- muscle pain
- malaise
- hypersensitivity
- sleep & temperature disturbance
- cardiovascular symptoms
- digestive disturbances
- visual problems
- vocal/muscular limitations.

ME is a very serious illness even in relatively mild cases. Research has found that ME-patients experience loss of function that is devastating and comparable to AIDS and late-stage cancer.

ME has a prevalence of 0.4% of the population with many of the sufferers being children.

It is the major cause for long term absence from school for children. In the UK ME is five times more prevalent than HIV/AIDS.

25% of people diagnosed with ME may be severely affected, house-bound, often bed-bound, left with little help from the medical community, often made to struggle to obtain benefits and left to an uncertain and debilitating future.

ME is estimated to cost European economies billions of Euros every year.

ME is a multi-system illness and distinct sub groups have been identified and some treatments have been shown to be effective.

To establish more comprehensive treatments and cures for these and other sub groups requires investment in biomedical research. Yet no public funding of biomedical research is currently taking place in Europe so biomedical research projects are funded solely by the private grants to individual researchers and from ME support groups and individuals.

With little funding of biomedical research into ME within Europe the EMEA are hoping to attract more support for research activities and hope to convince governments to recognize the necessity for a European biomedical research strategy to cure this illness.

ME needs more awareness from the public, politicians and healthcare staff.

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The European ME Alliance

The European ME Alliance has invited other organisations across Europe to support their objectives to change the perception of this illness and force change in government policies and accept the urgent need for biomedical research into the illness in order to establish treatments and cures for this devastating illness.

Member organisations of EMEA have agreed the following principles –

- Members of the European ME Alliance endorse the principles of the 2003 Canadian Consensus Document for Diagnosis and Treatment for ME/CFS.
- Members of the European ME Alliance endorse the principles of the 2006 paediatric definition from Dr Leonard Jason et al.
- Members of the European ME Alliance promote the fact that ME (myalgic encephalomyelitis) is a neurological illness in the World Health Organisation's International Classification of Diseases.
- Members of the European ME Alliance understand the necessity to use the composite term ME/CFS at the moment for ease of reference/standardisation.
- Members of the European ME Alliance support biomedical research into establishing sub groups of ME/CFS which will lead to treatments and cures for this illness.
- The European ME Alliance has, as an objective, the preparation and promotion of a common set of documentation, in all languages, for Alliance use that is supplemented by local information.

The founding members of the European ME Alliance are -

Belgium	**ME-Patientenvereniging**
Denmark	**ME-NetDK**
Ireland	**Irish ME Trust**
Germany	**Fatigatio e.V.**
Norway	**Norges ME-forening**
Sweden	**Riksföreningen för ME-patienter**
UK	**Invest in ME**

More details will be available in the coming months on the web site at www.europeanmealliance.org

or

www.euro-me.org.

ME Story

I've been dismissed, ridiculed, had so called medical professional try to humiliate me. I've had friends and family turn away from me. I've felt alone, been alone. I've felt depression, frustration, despair and anger at the way I've been treated over many years.

And I've seen how the attitude of the medical profession changes completely when one of their hallowed tests comes back with a 'positive' result.

All it took for me was the great good fortune of finding one doctor who listened to her instincts, that I was genuinely physically ill, and who persevered in trying to find the cause of that illness regardless of how elusive.

- Jim

Invest in ME announces the 2009 International ME/CFS Conference and continues our commitment to presenting the best knowledge, experience and research from the leading experts on ME/CFS. The conference provides an opportunity for researchers, healthcare staff, support, educational professionals, ME support groups and people with ME and the media, to hear the most relevant science, research, information and news on ME/CFS to be heard.

Our 2009 conference takes place on **29th May 2009** in London.

More details will be announced during the coming months so please visit our web site.

Contact: meconference@investinme.org.

Invest in ME

Energising ME Awareness and Biomedical Research

Welcome to London

We believe it is important to provide a possibility for people within government, health departments, social services, education and the media to be able to be informed of the the status of research, treatment and information related to Myalgic Encephalomyelitis.

Invest in ME offers the chance for researchers, medical practitioners, healthcare staff, people connected with, or interested in, the care of people with ME to present at the conference. We again hope to provide platforms for the following -

- Epidemiology
- Diagnosis
- Pathology
- Management and Treatment Protocols for ME
- Research
- Nutrition
- Care

The conference will again highlight the need for empirical evidence based on valid, modern and scientific diagnostic and treatment protocols. The conference will provide a chance to hear the latest news on ME from the most prominent speakers within the ME community - in ME Awareness Month 2009. Visit the conference web site home page at -

http://www.investinme.org/IiME%20Conference%202009/IiME%202009%20International%20ME%20Conference%20Home.htm

ME Conference Comments

"…thanks for organising a conference with such impressive speakers & at such reasonable cost. As a humble parent, most conferences are completely out of my price range, so was really delighted to be able to attend. I picked up lots of info & have realised that I need to do loads more h/w to really be on top of all the stuff that's been discovered since my daughter first became ill – 10 yrs ago." – Helen

"Many thanks for the wonderful conference. It was a great atmosphere and very uplifting to know of the wonderful work and people involved in helping us ME Sufferers. … It was a conference of excellence and it honoured us as well as raising us up!" – Jane

"I profited so much, I learned so much, I've met so many people I haven't met before - all this was so impressive." - Regina

"I thought it was fantastic, massively informative, encouraging, inspiring, necessary. It was very powerful hearing so much material from the doctors, researchers and speakers themselves, very, very impressive. I do agree that the speakers all came across as deeply humane. As a patient there was an enormous amount of useful applicable material and info on research hot from the lab so to speak. " – Nikki

See other comments at http://www.investinme.org/International%20ME%20Conference%202007%20-%20review%20feedback.htm

Plus ça change, plus c'est la même chose
"The more things change, the more things stay the same"

By Dr Bruce Carruthers

Dr Bruce Carruthers

Bruce Carruthers held an internship at the Charity Hospital of Lousiana, New Orleans, residencies in the Internal Medicine at the Hospital of the University of Pensylvania, Philadelphia, research fellowships at the American Diabetes Association in Philadelphia, and at the Clinical Investigation Unit of Shaughnessy Hospital, Vancouver.

He also had a fellowship of the Royal College of Physicians and Surgeons of Canada - specialising in Internal Medicine - and was a Research Scholar of the Medical Research Council of Canada.

He has specialised in diabetes and metabolic disorders and continuing clinical research in cellular information processing, diabetes mellitus and metabolic problems with a special interest in chronic fatigue, chronic pain problems of soft tissue origin and health enhancement.

From 1999-2003 he was the principal author for Canadian Consensus article 'Myalgic Encephalomyelitis/Chronic Fatigue Syndrome: Clinical Case Definition, Diagnostic and Treatment Protocols' which was published in Journal of Chronic Fatigue Syndrome 2003, 11: 7-115.

Until the present day Dr. Carruthers has continued to follow research interest in the role of consciousness in the clinical activities of Diagnosis, Prognosis, Treatment and Prevention. He produced in 2005 Myalgic Encephalomyelitis/Chronic Fatigue Syndrome : A Clinical Case Definition and Guidelines for Medical Practitioners - An Overview of the Canadian Consensus Document.

Dr Carruthers presented at the IiME ME/CFS Conference in 2006 in London (available on DVD from Invest in ME).

In the way that aphorisms have, the above saying describes a struggle between complementary attitudes towards reality that has been ongoing at least since around 500 B.C. in a disagreement between the Greek philosophers Heraclitus, who said the reality was change, and Parmenides, who said that reality was unchanging. This aphorism emphasizes that while being mutually exclusive by definition, the two approaches are both necessary in practice. The practice of medicine is guided by many aphorisms to reflect the complexity of the many complementary approaches essential to proper clinical decisions, which, while remaining mutually exclusive, are both necessary (1), including this aphorism.

The practice of scientific medicine also embodies this complementary struggle- while searching for the invariant laws of nature responsible for the mistakes of nature in the form of disease and dysfunction (contra-natural), it changes all the time while remaining complementary to the practice of clinical medicine which, while observing the vagaries of an individual's anecdotal experience of disease and

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dysfunction, has remained continuous throughout the time since Hippocrates, and undoubtedly before, since the essential situation has remained the same- a sick patient being tended to by a healer.

At the time of Hippocrates, there were 2 adjacent medical schools in Cos and Cnidos, each of which emphasized different approaches to handling the archetypal situation of a clinical patient (Klinikos (Gk meaning bed), presumably with a physician attending to an individual non-ambulatory patient more seriously ill) (2,3). Both types of physician dealt with symptoms, but one group took the nominalist stance that is all they had to deal with (presumably based on the assumption that symptoms are a natural prelinguistic form of language), this is the stance that symptoms have no intentional reference - that is they were not about anything but themselves- and should be dealt with at that level, by "symptomatic" remedies. The opposite realist position is that symptoms tell you about disturbances in an underlying causal reality which you have to learn to interpret properly. Both schools took the distinction between appearance and reality seriously, but the Cnidians felt that the appearance was the reality (nominalist) the surface symptoms were the level to address.

They analyzed symptoms as entities in themselves exhaustively, directing their therapies at what we call "symptomatic" measures rather than at any underlying cause of the symptoms. The Coans emphasized that symptoms were the surface appearances of an underlying unmanifest causal reality, towards which therapy should primarily be directed in the form of remedies and regimen to affect the humours, which names the dynamical causal forces they expected to be involved. The Cnidians emphasized a diagnostic search for static symptom clusters (what we now call syndromes) which were readily apparent to the observer, and could be studied as entities in themselves as to incidence, arrangements, etc. and could be examined by what they considered to be scientific methods (presumably those of Aristotle, since Aristotle's father was a prominent Cnidean physician, and somewhat similar to our natural history). The Coans, including Hippocrates, emphasized a method of prognosis, the real time search for evidence for less accessible underlying dynamic causal processes which they took to be causing the symptoms, (a realist position, which is also favoured by modern scientists and over which a recent war of attitudes has been fought, called the science wars (4). The realistic attitude certainly drives most research that is necessary to discover the causal network underlying ME/CFS, but given the current strategy prominent in the UK to emphasize a nominalist, at least on the surface, using a static "research" definition to discourage causally directed research and instead empiric methods to study (and also to promote and later institutionalize) nonspecific acausal therapies based on Cartesian body-mind dualism, one wonders about their motivation (see 5).

Unlike the NICE strategy, the prognostic search for evidence of underlying cause in individual patients is essentially dynamical, emphasizing change in the symptom severity and configuration as evidence for change in the underlying causal network that is assumed to be underlying these surface manifestations. In this approach any changes in the surface symptoms are assumed to be due to changes in the underlying causal network, and not in the symptoms themselves. This leads to an ambiguity in the assessment of clinical results, since symptomatic remedies can mask underlying causal change, and therapy directed against the underlying cause can result in symptomatic relief as part of the therapy. Note that the evaluation of both surface symptomatic and deep causal therapies depends on a reliable estimate of

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"The more things change, the more things stay the same"

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symptom severity and its changes over time.

This struggle in attitude has also resulted in two distinctly different approaches to the significance of syndromes or clusters of symptoms, a concept first used by Sydenham in the 17th century (6,7). A statistical measurement of symptom clustering will characterize it numerically, but cannot give any immediate clues as to the cause of this clustering. The idea embedded in so-called "research definitions" of CFS/ME is to establish symptom clustering by numerical measure, but to leave the search for causes for later science to decide. But what if that does not happen? We can act on the assumption that a cause will be found or that it will not be found, or that too many will be found each of which contribute uncertain force and relevance to the individual illness depending on its type (linear, circular, immediate, delayed, permissive, helping, enabling, allowing, formal efficient, final, pragmatic, etc) with the causal network assumed to be simple linear or complex and nonlinear, but with the whole situation certainly confused and uncertain. This will leave the diagnosis of syndromes in a limbo state, suspended between those that are expected to be caused somatically, and thereby explained, and those that are expected to be somatically unexplained, thus somato-form (having the form of somatic diseases but not the causal content) or caused mentally, "ín the head" as a default assumption. This indeterminate causal state arises when one follows exclusively a Humean type of perception, the acausal presentational immediacy which defers the question of cause and, in Whitehead's description, avoids the direct perception of "causal efficacy" (8). Hume's strategy avoids the immediate causal question of why these immediately perceived symptoms have clustered into a syndrome until later, to be decided by science plus inference, (presumably using the prospective, controlled observations of scientific experiment), with presumably more authority. However, as Montgomery has stated, working oncologists have estimated that they spend about 5% of their time using science to solve their problems in clinical judgment, and the rest doing "common-sense" (1, p 164). Aristotle already knew that science was not able to handle individual situations alone, and that is what clinical medicine is all about. While being informed by the general knowledge of science, a clinical judgment must be made using "phronesis" or practical wisdom(1,pp 33-41, 9 pp57-60), which is about unique situations, more or less comparable, and comes from a different sort of knowledge that allows the first person observations of the individual patient to be extended by the second person observations of the patient/ doctor while also bringing them together to interact with the third person general knowledge of science. This last interaction requires a fourth type of explanation that has been called paradeictic or pattern matching is used to bring first person and second person observations governed by deictic coordinates into interaction with the third person knowledge of apodeixis (10).

So what is this common sense? And why is it used so often, when scientific knowledge is so much better? It is because it is how we have all have learned about "how the body and the world works", i.e. the causal efficacy of its structures, based on the direct perception of the dynamic patterns of activity continuing since our babyhood. The implicit assumption that every felt effect has a cause which can and should be sought for is known as essentialism(11). It has been used by all of us since well before we could articulate anything like a theory of realism and is also expressed in the protolanguage of pain, fatigue, sleep, attraction , avoidance, smiles, cries, sneezes, coughs, nausea, anorexia, etc. The direct (anecdotal, uncontrolled) perception of causal efficacy has been demonstrated experimentally by and indirectly by Talmy as described in 12) who has introduced the

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concept of "force dynamics" by describing the many words and phrases found in naïve speech used to describe various types of causal relation which have presumably been observed directly in the "common-sense" world. Perhaps the clinicians have been using common sense methods to directly observe the causality lying beneath to explain their patients' symptoms, such as the direct perception of causality and force when the foot contacts a rock- did the foot kick the stone or the stone hit the foot? Samuel Johnson did not have to wait for RCTs to make his decision about the reality concerning forces and hard objects which are put into play when one kicks a rock, since he knew about intentional action. This common sense direct perception of cause and effect has the advantage of being applicable to the individual person in action, not indirectly inferred from a general group of Samuel Johnsons. The former anecdotal evidence is nonconfirmable from a scientific point of view, and thus to be scorned in some quarters, but essential to the clinician who deals with individual patients. It provides the essential contextual background from which an individual patient's symptom dynamical pattern arises to decide on what kind of cause and what is its cause, what are the effects and their severity, and whether and how these in turn become causes. Thus one can indirectly through dialogue, and directly, through examination and past experience, characterize the causal forces that lie beneath any symptom cluster, and thus become realists.

Clinical admonition;- Listen to your patient. He/she is telling you the diagnosis (?Osler see ref 26)

This ancient but ongoing clinical struggle involves 2 complementary strategies- 1/ Observing patients as individuals using dynamical, prognostic strategies to obtain reliable local knowledge directly in the local context of here and now (deictic) coordinates, which are thus changing all the time, and 2/ Obtaining general and (mostly) unchanging knowledge based on invariant laws of nature, using universal unchanging coordinates (apodeictic) to make a static model with decisions based on the group results. These results are assumed to be applicable to the individual patient (as long as he/she is not an "outlier"). The verdict of history seems to be that the Hippocratic approach has been more viable despite periodic attempts to re-instate a Cnidian approach to clinical medicine by focusing on surface symptoms and delaying or neglecting the search for underlying causes (for recent efforts besides those of NICE see DSM strategy towards psychiatric illness, which deals with symptom clusters and not with underlying causes(13). A similar nominalist approach is seen when research definitions are used to block research instead of facilitating it, holding on to cause-deferring research definitions well beyond their time. This subverts the proper function of clinical definitions which is to facilitate the identification of underlying general and particular causes in individual patients. Properly used, the general confirmed knowledge that is obtained from science is immensely helpful, but only when it is used as an aid in making adequate clinical judgments, instead of as a substitute for this local individual anecdotal clinical knowledge. Together general scientific knowledge and local clinical knowledge complement each other if used skillfully to cover each other's deficiencies.

The Canadian definition of ME/CFS (14) considers cognitive fatigue to be a member of the "neurological/cognitive" component, necessary to the case definition. It makes a huge difference whether one regards fatigue as a decontextualized, separated entity (see 17) to be included as one member of the numerical cluster of symptoms constituting an acausal syndrome (any interactive causal

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explanations to be deferred until later when we get our RCTs done a few years from now), and if one regards a syndrome as a dynamical pattern, a group of symptoms which constitute the surface manifestations of an underlying causal network or natural kind, the interactive forces of which can be directly felt implicitly even when not observed explicitly. In order to 'see/feel' this causal background one must not only observe just the surface manifestations but also feel the causal forces that underlies the surface. These disturbances are what the symptoms are 'about', their intentionality. If one separates the symptoms from what they are about by enumerating them, one decontextualises them (17), separating them from their usual causal background. Only if one observes the symptoms as they arise out of their dynamical background, can one feel this causal connection directly. Thus one must not only observe the symptoms when completed, but as they arise from the flow-of- life context in a prospective temporal dynamics, headed towards the future. If these are symptoms observed only by the patient themselves, in a first person perspective, the outside observer can only learn of their causal background by questioning the patient concerning the circumstances of their emergence, maintenance, aggravation, remittance, etc., but also by empathizing with her/him as an undivided whole and questioning/relying on their description of symptoms and their context within the developing second person perspective of the doctor /patient relationship. This is prognostic and direct observation of the clinical course of illness, a dynamical observation which has been emphasized since time immemorial to be at the core of the clinical situation. It is applicable to individuals on-line as they live their lives and suffer their symptoms along with any concomitant deterioration of activities. This is not the numerical "prognosis" which is applicable to members of groups after the fact.

Other clinical practices have been disturbed by nominalist, static approaches to the clinical situation. These include estimation of the severity of illness as observed in real time by its impact on the life-world with its deictic, here and now, individual coordinates and not the timeless general coordinates of science or the time of pure succession required by algorithmic approaches. Without the on-line observation-in-context of clinical practice one cannot see the impact of illness on a patient's life flows, their concrete dis-ability, whatever the results obtained in situations which have been de-contextualized for the sake of "objectivity". Without prognosis it cannot directly and accurately measure the effects of therapy, nor choose preventive actions to improve both surface and deeper manifestations of their illness.

Let me emphasize that while I feel that the disease title CFS/ME refers to a complex but discrete causal process which causes chronic severe, disabling dysfunction of an essential but extremely complex self-regulatory system of which we are only studying the Humpty-Dumpty fragments that have been opened up to become amenable to the study the linear causation within which science can identify causation. This search is well worthwhile since there is always the possibility that we can find a pragmatic cause within the complex bodily function, where a bit of biological matter such as viral nucleic acid or a vitamin-like chemical or a nutrient or a protein (e.g Ribonuclease-L) or a cascade of protein reactions, or a genomic dysfunction is responsible for the bodily dysregulation, despite its ultimate complexity.

The difference between an individual event which is causally laden and occurring at a specific place in space and time and the

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general term which may name it, which is abstracted and thus stripped of its direct causal attributes to become a member of a class with its own attributes, is a crucial one, a difference that makes a difference. An event has causes, but a class has relationships (25, p74). A living person is an event subject to a wide variety of causes over her/his lifetime and is a member of innumerable classes. The process of diagnosis consists of fitting a specific pattern of symptoms and signs exhibited by an individual patient in process, on-line into the general medical model to become a case of----, and thus a member of a class, which is abstract, off-line knowledge, a scientific model of disease. (This process has been called paradeixis (10). While one adds a large number of relationships thereby (those within the relevant disease concept of the current knowledge base), one thereby strips the case of the immediate causal relations available to it as an individual where they are felt as forces incurred in the ongoing living process she/he is immersed in. It is expected that these causal relations can be inferred as a result of controlled observations of a sufficient number of members of its class (cases of------). However the individual cannot be studied in this fashion. As previously mentioned, it has been known since Aristotle's time that no science of individuals is possible. This is because the observations cannot be controlled by comparing them to other members of the same set under various conditions (as members of a set), but that as an individual she/he is incomparable, living in a unique situation. So, since all of us live our lives at least partly anecdotally, uniquely and incomparably and if anecdotal evidence is inherently unreliable, then how do we survive? By learning "force dynamics" (11,12,15) at a young age, so that we can compare events along the time lines of our lives along in the various kinds of causal relations we encounter enroute. We start out as babies pretty incompetent in the ways of the world (by adult standards), but learn to regulate the movements of our bodies, our minds and our environments in ways reliable enough to allow most of us to live our "allotted" lifespan. We learn how each of these regions of existence "works". This process of learning goes on throughout our lives and is refined by "experience" and also specific training and practices, by learning how to cope with many kinds of stressors, including illnesses and much more. This makes our actions more and more accurate and reliable as we gain experience before the inevitable "anecdotage" takes over.

I would like to discuss three aspects of current situation regarding ME/CFS which are pertinent to what we have been discussing.

1/ Research vs. Clinical case definition.

By the time he/she becomes a case, a patient has already been abstracted into a case. Research definitions of ME/CFS(16), which are there to select clusters of similar patients to facilitate research, often include as optional symptoms cognitive dysfunction and sensory overload, listed as separate entities without regard for any causal relations with other symptoms or from its background. In clinical definitions designed to facilitate the identification of ME/CFS in individual patients (14), the search for the pertinent causal background (which is always unique) is facilitated by suggesting the connection of symptoms with various possible subsystems (pathophysiological systems that the symptom may be participating in) and by describing the various dynamical features of the symptom including on the force of interactions with other symptoms and the environment. These forces are observed directly at the commonsense level, and do not depend on whether these causal forces have been demonstrated to be explainable scientifically. They are there or they are not there.

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"The more things change, the more things stay the same"

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While touring the U.K recently, I was visiting Berkeley Castle. It was full of visitors and a number of subgroups were assigned to a docent to inform us concerning all the sites, times and gory details of its historical events. Each subgroup was assigned a different route. Since Berkeley Castle is not that big, the paths of each group crossed 3-4 times during the tour. Another group had a very unhappy baby as a member who was crying most of the time, and whose noise was sometimes close, sometimes distant. I noted that the docent began to lose her ability to keep her docentic speech train coherent during the times when the crying was loud. While this may have been explainable as a normal level of interference, it became apparent that the docent's cognitive impairment was so bad that she had to stop talking until the other group left the vicinity with each contact. After a decent interval her narrative resumed- smooth and coherent- until the next meeting with this particular noisy group, when the cognitive disturbance repeated itself. This did not happen during contact with other quieter groups. These interactive effects reappeared consistently until the end of the tour (about 3-4 times). While one interruption may be explained by chance and/or other causal variables, not a consistent 4, and there was a palpable interactive causal force observable at the times of these interactions, even by myself as an outside but informed observer. It is a common symptom duplex of ME/CFS that sensory overload will aggravate what I call "cognitive fatigue"- fatigue as a dynamic event and not a constant defect. The interrelation between these 2 events (the baby crying and the cognitive fatigue), when repeated consistently, was enough for me to assign interactive causality, whatever her own knowledge was about it and the direction of causation was certain the noise caused the confusion). This type of event if noticed was felt as dynamical and not subjective (as felt by the adult and her observers) , since there were no feelings of fatigue associated with the deterioration of cognitive fatigue (as far as I know since I did not ask her). The crying of the baby was steadily vociferous, and showed no apparent fatiguing during our relatively short time of observed interaction. This is one of the problems with cognitive fatigue- it has the dynamics of fatigue, but is often not accompanied by subjective feeling of fatigue- unlike the fatigue accompanying musculoskeletal exertion, and may not be directly observed by the perpetrator. But it is a very specific and consistent inter-relation, often noted by the patient when she/he is asked about it, and whose causal nature is confirmed by prognostic observation- over the course of repeated interactions over time. This everyday causal relation between interior and environmental events is not often included in discussions of scientific causation. But it is very real, and does not need an RCT to confirm it. It is also important, since it is affecting her competence as a docent. The causal relationship is confirmed by its felt force and consistency over time. It is the correlation dynamics (18) that confirms the causal relevance/irrelevance of this connection between the 2 variables of a baby crying and a docent's cognitive dysfunction. It is a causal interaction depending on the loudness of the crying and the vigor of the cognitive system involved. The point of cognitive fatigue is that it is dynamical- her mind does not work well in the presence of the disturbing variable, but is fine under many other circumstances. I would expect it to be regarded as inconsistent if the observer is looking for static entity called "loss of concentration" exclusively, since it is not always there. Perhaps the patient cannot screen out other sensations to concentrate on what she is doing, but it is important for her to identify this specific interaction, since she could prevent it in the future by avoiding the situations where it occurs. Thus it will allow her to apply a specific preventive measure that she can learn. Unfortunately this type of

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causation is not usually included in descriptions of the etiology of ME/CFS since it is too "anecdotal".

Cognitive fatigue is often not picked up or is under-estimated during psychological testing because an ethical tester is supposed to throw away data obtained while the client is fatigued. This rule results in interpreting dynamical findings as inconsistencies. So here is the dilemma. A "fatigue", which by definition is dynamic- it comes and goes- is not accounted for in an objective thought system which requires objects, which by definition are stable and unchanging, and thus ignored. In such a system entities are not objects if they are changing, and thus to be discarded. If observed in a dynamical system which is set up to observe this coming and going, the fatigue would be very real and consistent. But to see this requires a prognostic dynamical approach. In such an approach the relevant causal events are not only in the body, they are in the world, (embedded, embodied dynamics), and they may shift with changing circumstances (causal spread- 17). While such an interaction could of course be studied in the standardized environment of scientific experiments, it can also be identified and confirmed in the on-line, 'wild', unique, anecdotal, situation described.

2/ Observations of "kinds" of fatigue to identify sites where causal patterns change.

Research into the development of mind in children has revealed that children are born "essentialists". What does that mean? Gelman (11) has given reasons for why children are essentialists, in their ongoing attempts to understand the underlying causal structuring of the world and of their own bodies. It works by learning to identify "kinds" of thing that do this or that so that they can learn to predict their own and others' behavior. 1/ they act as if they are aware from earliest infancy that there is an appearance/reality distinction, such as we have mentioned before. They learn what experts would call "induction from property clusters" or what additional properties to expect from a given cluster, especially homeostatic clusters that work to stabilize the organism and maintain its invariance through environmental change . They learn causal determinism, with its search for hidden, non-obvious, as-yet-unknown "natural" properties that can explain cause through inherent properties or essences. They also learn to track identity over time, thus becoming dynamicists and followers of both Heraclitus and Parmenides. They learn deference to trusted experts (starting with their parents) to fill out what they do not know, (and as a corollary, to avoid the opinions of those they have learned not to trust- my addition). In exploring the feeling they later learn to call "fatigue" they learn how it is causally connected with activity (they feel fatigued after activity and restored, more energetic, after rest), and learn to expect this dynamical relation and how the feeling differs from sleepiness and weakness. They learn that there are other kinds of fatigue- that of a different flavour (malaise) that they feel with a viral infection, or the fatigue they feel if they have not had enough sleep or if they have been on a long airplane ride, again with different characteristics or 'flavours' which can give causal clues when felt with discrimination.

This implicit knowledge about the causal relations surrounding fatigue is embedded in our experience of the varying contexts of daily life, which is a changing flow, not a static state. It is our concepts which have been abstracted from this dynamical state that are constant. In the resting state the body is felt (consciously or not) by the generalized sensation of proprioception, or self-perception, as the presence of an nonmoving body. When

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active, the body is felt more acutely by the same proprioception as a moving body, moving in a variety of dynamic patterns. Self-observation of this embodied and embedded movement, both inside and in relation to outside, is continued into the environment by the rods of peripheral vision, which are especially sensitive to movement and patterns of movement, and work faster than the complementary colour sensitive cones. Symptoms arise from this proprioceptive base as signals to modulate bodily activity in coordinatation with patterns in the immediate environment. Thus they form a coordination dynamics (18). Since proprioception is a reflexive sense, its truth conditions are of a different kind from those of the distancing senses. They are directly and reflexively felt (nondual), not as an outside observer separated from its object (dualistic). If bodily you feel fatigued, it is time to rest. If you feel energized with more potential to move, you may choose to become active. We have all learned about this causal flow and how to modulate it in relation to the environment since first becoming mobile in infancy. This learning happens semi-automatically, depending on our choice, training and circumstance.

What happens if this regulatory system becomes disrupted? What if fatigue becomes "delayed", and it becomes harder to causally connect the fatigue with the preceding activity. Fatigue does not follow activity in the expected causal rhythm, and the situation becomes confusing since there has been a change in the dynamics. "The goal posts have shifted", as I tell my patients. A new "attractor" or pattern of activity has been formed in the language of dynamical coordination theory (18). What do we do? At first we try to ignore the fatigue and get on with the work of our life despite the feelings of fatigue by "will power" or the use of stimulants. This is the King Canute strategy. What happens then? We "crash" as the fatigue gets worse. The more we try to overcome this fatigue, the worse it gets. It is this dynamical pattern change in the activity regulation system of the whole brain-body-world that we must address. It is addressed by pacing- adding your consciousness into this ordinarily semi-automatic regulatory system with added regulation using self-directed, non-dual mindfulness(19) that you can begin to connect the fatigue/ activity relationship which has been dissociated. With temporal dissociation from activity , the cause of fatigue is put into turmoil. It could be caused by numerous intervening variables, as it is not work in its usual rhythms. One cannot rely on these subconscious dynamic pattern any more. One needs to intervene with the conscious mind which can re-assert the connection between activity and fatigue if it is there, or search for other answers to the problem that has been posed by consciousness (using mindfulness). Then the pacing of activity can begin, however delayed the causal connection between activity and fatigue has become. While essential for the repair of your basic activity self-regulator, this conscious symptom guided proprioceptive system must learn to discern other kinds of fatigue with varying causal backgrounds- induced by lack of sleep, by intercurrent infections, cognitive overload, exposure to too much noise, light, odours, cognitive work, circumambient noise or busyness, stress, emotional overwork, toxic exposure, etc. This all gives you clues as to what is causing your present state of fatigue. These are the interactive commonsense causal factors coming in from the environment in which it is embedded with variable temporalities. Causal variables also come from the embodied inside. The symptoms of the syndrome will disturb the basic control system depending on how interactive they are. Better regulation of the whole activity/fatigue control system will not come from learning to ignore

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these signals in deference to regulation from the outside, using external rules, whether these be cognitive rules from a separated dualistic "mind" see (17) or from the outside as coercion. In either case this would have become an external control system, not a self-regulated one, and clumsy in its reaction to changing circumstances, thus less of a self-regulating system. But what you do need to do to modulate a self-organizing one is to become more mindful of your proprioceptive sensations, including your symptoms, where the observer is identical to the self-observing, self-organizing system.

If internal symptoms are very interactive, they should be regarded as part of the syndrome (e.g. if a patient becomes fatigued after having a bowel movement, this interaction should be regarded as causally relevant to the fatigue if it is severe and consistent enough to contribute to disability. If general pain, gastrointestinal pain, headaches, depression or anxiety become sufficiently causally interactive, they should be included in the syndrome. The point is to try to estimate how causally interactive they are, a measure that can only be done using the dynamical methods of clinical medicine with its deictic individual coordinates of "here and now". The ultimate cause of the disturbance is the dysregulation of a superordinate self-organizing system in its relation to its subordinate systems with both bottom-up and top down control. The ultimate cure is to re-establish proper bidirectional regulation. The dynamical difference between the delayed fatigue of ME/CFS vs. non-delayed normal fatigue implies a distinct change in causal network which underlies this change, and indicates that a causally relevant shift in the "kind" of fatigue is happening. It is not just a more severe variety of the normal kinds of fatigue. It is a distinctly different kind of fatigue, to be classified under a different taxon (20), thus implying a distinctly different causal pattern lying beneath its surface manifestations. The features of this dynamical shift, if paid attention to, can thus orient research to find the relevant cause(s) responsible for this shift in dynamic patterns without having to render the whole causal system explicit (which may be impossible, or complex enough to keep researchers busy for many years ahead). If one continues to ignore this search for dynamically different kinds of fatigue by using static decontextualized models of "fatigue" conceived as a static entity, this type of research will continue to be blocked.

3/ ME/CFS "fatigue" as embedded in a system which regulates the basic complements of activity and rest, and its comparison with the regulation of blood glucose level in patients with "brittle" diabetes mellitus.

Since "fatigue" by being considered as a nominal entity in isolation from what the fatigue is about- an altered bodily state- it will continue to be regarded as "subjective" feeling and thus not as an integral part of a regulatory system which must function orthogonally to the Cartesian type of subject/object split which is a prerequisite for our current scientific thinking to work. Fatigue is embodied, not floating around the subjective mind in an endless chain of cognitions. I will use the framework for a regulatory system first suggested by Ashby, a founding father of the dynamical systems approach (22). His model of the brain is that of a homeostat, a self-regulator. A self-regulator keeps "essential variables" within tolerable limits (unchanging) by changing itself in response to changes in the environment. The self-regulator is the part of the organism that changes in order to keep the essential variables the same. The essential variables must be kept the same if the whole organism is to survive. The changing part, the regulatory part of the system is designed to change on demand. The major parts of the organism that

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are allowed to vary in this way are the brain, the immune system and the endocrine system. If these systems are changing appropriately, the whole organism is healthy. Disorder or disease happens when their changes are not appropriate. Ample research has shown that the major self-regulatory systems are involved in the causation of ME/CFS (brain plus CNS and ANS, endocrine and immune systems). Additional research has indicated that the fluid and energy transport system, including the circulatory system and mitochondrial transport may also be seriously involved. What we don't know is how this all fits together and works as a self-regulatory system. It is only in the intact patient that we can observe or infer their various dysfunctions. We do not know if a single glitch can upset a lot of biochemistry (e.g. B12 deficiency, aberrant ribonuclease-L molecules, cytokine production, etc) with cure by replacement. But the whole control system that we must focus on is that regulating physical activity/rest, including the subjective feeling of fatigue as one of its essential parts. Dynamical disturbances of other sorts will be causally coordinated with other kinds of fatigue that may be implicated- cognitive fatigue, stress fatigue, immune fatigue, cardiac muscle fatigue, mitochondrial fatigue, acceleration fatigue, etc. While the overall effect of pathophysiology is inappropriate fatigue in a system designed to self-regulate overall activity in a dynamical and holistic system, any glitches responsible for this fatigue may be quite specific. Unfortunately the currently approved treatments for this condition in the U.K. (CBT and GET), are attempting to adjust symptoms using an external site of control. Thus these approaches ignore the crucial self-organizing aspect of any biological control system.

I would like to compare this situation with that of the system regulating blood glucose level, one implicated in the genesis of diabetes mellitus. Many patients with diabetes do not have a serious control problem. Their blood sugars may be too high, but the level is stable or changes slightly and gently. The diabetics who have a control problem are those that are designated as "brittle" because their blood sugars go up and down violently. Before the discovery of the hormonal protein insulin, the more severe diabetics mostly ran the higher blood glucose levels, and many died of diabetic acidosis. Pre-insulin physicians tried to regulate the glucose level using diet and fluid manipulations. (23) With the advent of insulin therapy, most diabetics did well, but a few remained "brittle" because they were sensitive to the insulin so that their blood sugars fluctuated up and down between tolerably high and low blood glucose levels. Because of the effectiveness of insulin therapy, new disease of hypoglycemia or low blood sugar became prominent. The biochemical system underlying this glucose control became more and more complex as problems of insulin resistance, anti-insulin hormones such as glucagon and cortisol were added to the control system as did allergic hypersensitivity to foreign insulin and contaminants of human insulin. With the new iatrogenic disease of hypoglycemia, a new system of anti-insulin hormones were engaged as the adrenalin release that a sudden drop in blood sugar caused became more important. Experienced diabetics could often consciously discern where their blood sugar was, by how there body felt when sugar was up, down, falling slowly or fast, etc. but this estimate was only rough. These estimates were originally vague but became more precise with experience. These symptoms depended not so much on the level of glucose, but on its rate of fall. Because of all these complexities, it has been a great boon for this type of diabetic to be able to measure glucose levels "on-line" as it has improved their ability to regulate their blood sugars immensely. However they have to fall

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back on bodily feelings when blood testing is not always available. It is obviously impossible to regulate these patients from afar (external site of control). These patients whether using blood sugar levels or their own internal feelings have to learn to self-regulate, and on a dynamical basis. Of all the potential variables in this complex control system, only 3 are essential- the diet, the dose and timing of insulin, and the exercise must be timed and varied on a frequent basis. The diet raises the blood sugar, depending on its type and amount and the exercise, (again depending on type and amount) and insulin lower it. Osler's 1914 textbook in internal medicine does not suggest using exercise as a regulator, since it wasn't yet a crucial control variable, but prescribes "modest exercise" (23). Timing and balancing these variables is an on-line dynamical process. While many other variables can intervene- e.g. rises in the anti-insulin hormones due to stress, infection and anxiety, inability to control due to cognitive problems secondary to hypoglycemia, etc, they are more like external parameters that affect the whole state of the control system than like variables within the system. Emotional problem are common, often during adolescence, when young diabetics during their rebellious adolescent stage will often try to ignore the illness as well as resist such intrusive and bothersome therapy. But it is obvious to all that these emotional disturbances do not cause diabetes, since this is obviously basically a physical kind of dysregulation. They are parametric (22, p71ff) aggravators, quite distinct for the control system itself, and can of either physical or mental kind. Any psychotherapy initiated to get the patient out of her/his adolescent rebellion could certainly improve the diabetic control, and yet not be regarded as a treatment for diabetes. The situation is clear.

I would like you to compare this situation with that of ME/CFS. There is no problem in assigning causal responsibility in diabetics. Why the problem? One is that a complex multicausal regulatory system involved with ME/CFS has proven to be a difficult "entity" to grasp through research. As one does more science, the whole system will undoubtedly become more complex, and we need to guide research towards regions that are likely to be fruitful. Another impediment is that a prominent strategy in current use to guide attitudes and treatment methods regards ME/CFS as a somatoform disorder, i.e. a symptom cluster showing the form of a somatic organic disorder, but without the content. This is actually more of a default position than a diagnosis, but is similar in that it is serving as the termination of a clinical judgment procedure. This has led to disagreement as to whether CFS/ME is a physical or a mental kind of disorder, and within the system which are its constitutive variables that determine its form and which are the parameters that influence it but don't determine it- with the different kinds of causality that this entails and the different forms of treatment to follow. The result is a mess. We have discussed above how the research definitions have been used not only to guide future research, but also to ignore current research findings until the story has been completely told and "the cause" of CFS/ME is known. If we have not been able to demonstrate the complete causal network with complete scientific certainty, should we continue on the assumption that there is no underlying cause for these symptoms and that they don't refer to anything except their nominal essence as a name, thus remaining a "somato-form" "nominalist" kind as a default interim position while we search for the underlying cause that fulfills the symptoms' intention as a "natural kind" (21). It is very tempting to slide from the attitude that a symptom's causal background is uncertain, to the attitude that it is not there, and hence that the illness is all symptom and no reality. From

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here it is easy to slide into an attitude that since by definition somatoform disorders simulate the form of real diseases, that the patients' experience is not to be trusted. Their illness has been moved from being regarded as a physical kind to a mental kind without giving them the benefit of the considerable doubt. This in turn entails large shifts in trust and attitude from other people and agencies, stigma of a regulatory disorder that have been assigned to the mental side and of the consequences of this- how patients are treated in general at all levels of supervision, the types of treatment offered and how it is administered (21). It interferes with the assessment of symptoms as they have been removed from a natural realm into a distinctly separate symbolic realm, the fruit of Cartesian dualism between mind and body and thus an ontological shift that many patients can feel proprioceptively as a nondual jolt in their second-person discourse with their physicians with immense practical repercussions.

Far more preferable would be to give the benefit of doubt to the patient and assume that there is an underlying natural causal network underlying the disease, of which we know fragments but not the complete story. In the meanwhile we should focus scientifically on the evolutionary development of symptoms, what somatic symptoms refer to and what has been the selective value to justify their retention? The biological function of these symptoms is to refer to their underlying causes in the interest of better self-regulation of a mobile organism living in a group. What is the system that they refer to? We should continue studying clinically the dynamic causal patterns that patients produce in their illnesses to identify sites where a shift in pattern makes scientific search in the region exposed more likely to be fruitful. It is a bit like searching for oil. Since these symptom patterns are discovered arising from the anecdotal experience of individual patients, the lack of anecdotal trust in patient experience has been aggravated by the current thrust towards "evidence based medicine" with its push towards exclusive reliance on general knowledge vs. particular knowledge without accepting a complementary relation between these different kinds of knowledge which are both necessary.

We should return to observing patient experience precisely as the symptoms, embedded in the flux of life, arise to out of their causal background in discrete dynamical patterns. In ME/CFS the dominant symptom of fatigue should be observed on-line by both patient and physician as it functions in the self-regulatory system of activity/rest modulation and look for the essential variables that stabilize the system so that the patient can learn to self-regulate it better on an ongoing basis. The interactions between fatigue and other symptoms should be studied for the causal efficacy that makes a syndrome into a dynamical causal entity. These should be distinguished by their dynamics from the external parameters that affect the state of the as a whole, stabilize it, destabilize it, change its dynamic form, etc. In studying these dynamical details it will also be helpful to search for "homeostatic clusters"(24) which are crucial for steadying the system.

These studies can be expanded to as examination of how the individual organism can stay self-regulated in the larger social and cultural environment despite the impingement of external regulatory forces which exert greater forces and work according to a different dynamics and undergo parametric change all the time. But this will need yet another study- the world goes on changing, and we stay the same by changing with it.

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A Severe ME-aware nursing model

By

Greg Crowhurst RNLD , PgDip Experiential Learning , Cert Counselling Skills, MA

This article outlines a self-reflective nursing model , in order to enable practitioners to enter into a sensitive partnership with patients who have severe Myalgic Encephalomyelitis / Chronic Fatigue Syndrome.

There is an urgent need to develop an appropriate model of practice for people with Severe Myalgic Encephalomyelitis (ME), if practitioners are to avoid tragedies like that of Sophia Mirza, who died from ME , after suffering appalling treatment at the hands of doctors and nurses following sectioning under the Mental Health Act for two weeks in 2003.(Hooper 2006).

Crawford, Aitken and McCagh (2008) recently found that nurses still respond more positively to patients with Multiple Sclerosis and Rheumatoid Arthritis than patients with ME/CFS, which they are more likely to wrongly view as a psychological disorder. Nurses also report low levels of training and confidence in their skills when working with patients who have ME/CFS.

A great deal of conflicting advice still surrounds ME/CFS , leaving many patients "dismissed and abandoned without support. (Hooper et al 2005).

Central to the care of people with ME/CFS and the cornerstone of any nursing model (Archibald 2000) are the beliefs and values, the experience and knowledge of the nurse.

Background

There are an estimated 62, 500 people with severe ME /CFS in the UK (DH2002) .The disease, which can occur in both sporadic and epidemic forms (Jenkins 1991) has been described in the medical literature for about 70 years. Over 4,000 papers have been published, documenting the biomedical abnormalities found in ME/CFS (CDC 2006)

Greg Crowhurst cares for his wife, a long-term severe ME sufferer.

A comprehensive series of video by the author , showing the impact and the reality of severe ME , are available for free online at: http://www.youtube.com/user/gregcrowhurst

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Since 1969 ME/CFS has been classified as a neurological disorder by the World Health Organisation . ME/CFS was recognised as a specific disease entity by The Royal Society of Medicine in 1978 and as an organic disorder by the Department of Health in 1987 (Hansard 1987).

Included in the NHS National Service Framework (DH 2004) as a long-term neurological condition, cycles of severe relapse are common in ME/CFS as are further symptoms developing over time. "Substantial improvement is uncommon and is less than 6%" (Anderson et al. 2004); and, "Full recovery... is rare" (Cairns & Hotopf, 2005).

The Experience of Severe ME/CFS

It is not 'fatigue' or 'tiredness' that is the one essential characteristic of ME/CFS but central nervous system (CNS) dysfunction (Bassett 2006).

Bell (1995) describes the word "fatigue" as: 'A very inappropriate term for what patients experience. It's not really fatigue at all, which

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A Severe ME-aware nursing model (*continued*)

is defined as a normal recovery state from exertion and that is precisely what does NOT happen in this illness"; this is extremely important for the nurse to assimilate, in order to effectively work with people with severe ME.

Everyday life for the severe ME sufferer is a perpetual struggle. As Owen (2007) points out the most severely affected may not be able to speak, eat, swallow, open their bowels. They may not be able to sit up or move themselves, they may be too exhausted to dress or wash . The sound of running water may be too much for them to bear, they may not be able to open their mouth to brush their teeth.

Crowhurst L. (2007) describes how : "*Having severe ME is unimaginable ; the experience is so different , intense and unremitting than anything I have ever experienced before. I am never unaware of the range of symptoms that rage through my body , and are dominated by intense never ending pain in every millimetre of my skin and muscles, over and throughout my whole body; head shoulders, back, front , arms legs, hands , feet, toes , fingers, eye lids , scalp the soles of my feet, the tip of my nose , my eyebrows even. They all burn, throb, tingle, itch, and hurt in ways indescribably unbearable , along with other unusual sensations*"

There are no known appropriate treatments for ME/CFS available at this time and it has been found that some of the mainstream therapies applied to ME sufferers have been unhelpful or harmful on many occasions , especially treatments such as Cognitive Behavioural Therapy and Graded Exercise Therapy.. (Crowhurst 2005).

Knowledge, sensitivity and awareness are paramount. The nurse must be able to respond creatively in order to aid the person. This means learning to understand:

- what is needed,
- when it is needed and
- how it is needed ;

which may not always be obvious or repeatable.

Any activity where thought and action work together can easily become out of reach of the person with severe ME/CFS, without any clear understanding or explanation of why. Simply speaking on a telephone for example, could be far too much for the person :

Management :
Family members and carers, with the patient's agreement , can contribute a wealth of essential knowledge and valid information, in the development of an individualised management plan.

People with severe ME/CFS are at great risk, generally, of a dramatic increase in their symptoms, which could plunge them into even greater depths of illness ; this is especially so if

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Facts About ME

'It is accepted by the most experienced ME clinicians that some degree of encephalitis has occurred both in patients with ME and in those with post-polio syndrome: the areas chiefly affected include the upper spinal motor and sensory nerve roots and the spinal nerve networks traversing the adjacent brain stem (which is always damaged).

In nearly every patient there are signs of disease of the central nervous system.'

- Professor Malcolm Hooper

A Severe ME-aware nursing model (*continued*)

The person needs to :

- Be able to hold the phone or put on a headset.
- Be able to bear the noise.
- Have the energy to physically answer the phone.
- Have the energy to speak.
- Have the ability to focus.
- Have the ability to concentrate.
- Have the cognitive ability to receive the information.
- Have the cognitive ability to process the information.
- Have the emotional strength to deal with the other person's emotional state.
- Have the ability to cope with the tone of voice, loudness of voice , pace of conversation..
- Have the ability to access information if they are asked a question.
- Have the stamina to cope with a conversation of unknown length.
- Have to ability to cope with waiting in a queue or waiting for a person, which uses an inordinate amount of energy and they may run-out of ability.
- Have the ability to cope with the increased symptoms that will follow having used the phone.
- Have the ability to cope with the potential shutdown of various systems that should support the actions they are doing, for example, their muscles running out, noise becoming too loud, their voice going, while on the phone.
- Have the ability to cope with the post-exertional impact. (You have to remember that physical and emotional energy are equivalent in ME/CFS .)
- Have the ability to coordinate their thoughts, energy and physical ability. the more complex the task, the more impossible daily living becomes and the more isolated the person becomes from the normal world; because the normal world becomes out of reach because of the complexity of the tasks and the impact of the multi-system dysfunction of the body.

The difficulties of speaking on the telephone for the severe ME/CFS sufferer.

they leave the safety and security of their known environment :

In planning any transition a host of environmental issues need to be taken into account well in advance, for example :

- **Physical comfort**
- **Weight of bedding**
- **Softness of mattress**
- **Neck and back support**
- **Noise and light exposure**
- **Food /chemical sensitivities/allergies**
- **Timing of meals**
- **The how and when of physical assistance**

The nurse may not have the answers, but it

(continued on page 36)

A Severe ME-aware nursing model (*continued*)

ME/CFS is characterized by (Mark 2005) :

- malaise following even modest physical activity
- delayed reaction to physical and/or mental activity (up till 24 hours and more);
- abnormal length of convalescence (out of proportion to level of activity)
- varying and fluctuating symptoms during the day, but also in the course of days, weeks and months

is very important to ask the right questions and not assume anything, as this Observation and Assessment tool shows (see Table 1) -

As the chart shows, in severe ME/CFS sleep/awake times and personal care needs are unlikely to fit into standard patterns.

An ME-aware Nursing Model

In the author's experience (Crowhurst 2005) , the most appropriate nursing approach is one that incorporates the Nursing Process (Yura and Walsh 1967) within a self-reflective model of practice .

Crowhurst (2005) has outlined how the experiential learning cycle (Kolb and Fry 1975) can be used to underpin ME/CFS nursing practice , encouraging nurses to reflect upon their practice experientially and holistically. Some important areas for practitioner reflection are listed below in Table 2 :

An ME-aware approach requires the nurse to be :

- particularly conscious of how ME/CFS manifests.
- the full range of symptoms.

(continued on page 38)

Table 1 - Severe ME/CFS : Observation and Assessment tool

Symptom	Questions/Observations	Comments
Hyperacusis	What is the patient's response to electrical equipment, noise, telephone, doorbell, washing machine, Hoover ?	Noise sensitivity can be so great that even a whisper sounds like a shout; it may be painful and it may increase a whole range of symptoms.
Hyperesthesia	Does the patient flinch, become irritated and depressed ? Is the skin hypersensitive to touch ? May be unable to tolerate massage, stroking, accidental contact.	The patient may find any kind of contact or movement over the skin unbearable. May flinch, may react strongly, verbally, be very distressed by even a slight brushing. The nurse has to be very careful and aware.

(Table 1 continued on page 37)

Pain	Is the patient experiencing sleep difficulties because of pain ? Do they need special aids and equipment ? Are there analgesics that help/ease the pain? Is touch and lifting difficult because of pain ?	The patient with severe ME might experience muscle pain, nerve pain, skin-crawling sensations, burning, itching, throbbing pain. The person with ME might feel extremely ill al the time, on top of the other symptoms. It may help to identify some of the symptoms in order to aim for relief.
Multiple chemical sensitivity	Does the person feel nauseous, experience headaches, rashes or other symptoms in response to being exposed to certain chemicals, smells, perfumes, toiletries, household cleaning agents ? Have they developed specific food sensitivities/allergies ?	The nurse must be aware that perfumes, deodorants, might have a deteriorative effect on the person with ME, which can be extreme and immediate. Household cleaning agents etc require careful consideration. Organic products might be less harmful.
Orthostatic intolerance	The patient may become greatly distressed moving from lying to sitting, to standing. They may be unable to sit upright. They may experience dizziness, increased feeling of illness, panic even, if made to stand.	The severe ME sufferer may feel utterly ill and/or unable to stand, but may not be able to identify why. It is important for the nurse to know there is a physiological reason for this.
Unrestorative sleep	Does the patient feel more ill and in more pain upon waking ? Do they feel unrefreshed and unrested ?Do they have difficulties going to sleep and staying asleep ? Do they have difficulties waking up ? They may need to sleep during the day ? Sleep may push the person into a worse state of illness and paralysis.	The sleep pattern in ME is altered. May be awake during the night and asleep during the day. They may not experience restorative sleep. They may have nightmares. Paralysis is a significant symptom in sleep disorder.
Muscle dysfunction	Can the patient do something one moment and not the next ? Does the person have difficulty holding implements, difficulties with gripping ? Do they have difficulty holding even a light object ? Do they have difficulty sitting or standing at varying times during the day ? They may require help eating, or vary in degree of help needed.	The ability to use any muscle may come and go and vary throughout the day and night and is beyond the control of the person with severe ME. They may be physically able to do something one moment and not the next. The patient must never be pushed to do something, just because they can it sometimes. There is a post-exertional malaise response to using muscles, that can occur up to hours and days afterwards.

A Severe ME-aware nursing model (*continued*)

- the likely impact of any interaction upon the person.

The prepared nurse can greatly lessen the chances of any deterioration in symptoms :

ASSESSMENT :

It is the way the nurse approaches the patient with severe ME/CFS that determines the outcome of assessment.

Particularly with ME/CFS, the underlying beliefs, knowledge and understanding the nurse has about the disease itself, could result in two very different assessments, depending upon whether the nurse believes ME/CFS is a physical disease or a mental health issue and whether they adopt an authoritarian "I know best" approach or a more empathic partnership style of relating.

Not only are the nurse's views and understanding of the illness important, their awareness in regard to their own power and responsibility are vital. An authoritarian approach, coupled together with an assumption that ME/CFS is not a real disease, that somehow the "patient is just not trying hard enough" , or has in some way caused their illness by wrong beliefs, means that the assessment will be deeply flawed.

PLANNING :

Again, the nurse's underlying assumptions and knowledge of the disease will play a crucial role in planning any intervention. Unless the nurse is aware that the person with ME/CFS is a long term chronically-ill patient, who is unlikely to get better (anyone severely affected for more than 5 years has a poor prognosis of recovery (DH2002)) , the planning may be way too hopeful with way too high an expectation of the patient. The nurse's preparation must be done with awareness before intervention in the patient's life.

Planning should be focused upon the way the nurse interacts and responds to the patient. Without key- planning the dangers of an immediate worsening of illness and a long term relapse through poor understanding are likely outcomes.

IMPLEMENTATION :

Implementation needs to focus on acute awareness of the severity of illness and the multi-system dysfunction the person is experiencing. This cannot be emphasised enough or over-played ; it is the key to any successful intervention. The nurse must be able to be flexible and understand the potential impact of any movement, speech, action, upon the severe ME/CFS sufferer and must always trust and listen to the patient and their reaction.

The response of people with ME/CFS is not always predictable; often the opposite rather than the expected occurs. This must be understood by the nurse . It would be well to consider alternative interventions in advance, so the nurse is prepared when something is not working.

Even if an intervention is not possible at one moment, it may still be possible at some other point in time, for there are fluctuations of experience of symptoms within the general chronicity of the illness.

EVALUATION :

Integrity, wisdom and patience are required. Any improvement or response may be extremely small, almost invisible perhaps to the nurse, yet the person with severe ME/CFS may discover significant benefit from what might seem like a small, even insignificant outcome. Patience is particularly required, both for the nurse and for the patient.

(continued on page 39)

A Severe ME-aware nursing model (*continued*)

Table 2

MIND : • What am I thinking about when I approach the severe ME/CFS sufferer? • Can I focus solely about what I am doing ? • Have I thought ahead about what potential issues might come up ? • Do I understand that ME/CFS is an organic, physical disease ?	**BODY** : • What is my intended posture ? Open ? • Partnership ? • Is my physical posture in keeping with my intention ? • Am I able to be gentle enough , when I help the patient ? • Am I too tired to help sensitively and carefully ? • Am I in pain anywhere myself ?
SPIRIT : • How do I feel about being with the patient ? • Can I connect with the patient and their need ? • Am I flowing with the right energy to make contact with the person ?	**EMOTION :** • What is my emotional state ? • Is it going to have a good impact upon my interaction ? • Am I distracted about other issues ? • Am I distressed by the patient's issues ? • Do I feel good about myself ? • How do I feel about the patient ?

Because of the severity and the long-term nature of the illness and the ease with which any intervention can lead to a worsening rather than a bettering of illness, how the nurse approaches an evaluation is very significant.

It must be remembered that the person with ME/CFS may have severe cognitive dysfunction, may not be able to write or read, speak, understand or cope with questions. A very gentle approach is essential in developing a partnership with people who have ME/CFS.

An example Case Study (see Table 3) :

(continued on page 40)

ME Story

Now, nearly eight years later, going from a student at the top of my class with an unlimited future to a dependent, rather helpless person with no real hope for healing is something that only others in this situation can understand. Meeting new people, and having them ask, "what do you do?" makes me cringe. The amount of shame and isolation at times is unbearable, but there is also a glimmer of hope that with greater understanding will come better treatments or at least compassion.

- Jessica

A Severe ME-aware nursing model (*continued*)

Table 3

Ms H is 50 year old woman with severe ME .

Nursing intervention : assisting patient with eating lunch

Assessment

Ms H's main symptoms are :

Symptom	**Impact**
Pain :	cannot bear touch
Transient paralysis	unable to use limbs affected.
Numbness	cannot feel properly
Muscle weakness	cannot grip properly
Muscle fatigueability	post exertional malaise/pain increase
Light sensitivity	needs dark glasses, low light
Noise sensitivity	any noise can hurt and aggravate symptoms
Food sensitivity/allergies	can only eat certain foods, no dairy, wheat or oil
Gastritis	stomach pain, bloating
Hypoglycaemia	irritability, distress, worsening symptoms
Swallowing difficulties	food gets stuck
Breathing difficulties	malaise increases, chest muscle pain
Spasms	head, limbs, body shaking violently,

Presenting Issues :

Variability of symptoms and severity

Functional difficulties in eating

Potential deterioration to complete incapacity

Sitting up/postural issues

Planning :

Intention :

Provide finger food

Provide padded seating and back/neck support

Low light

Quiet, peaceful environment

Ensure appropriate diet

Provide assistance with drinking (straw).

To have a warm open accepting posture with no quick movements, minimal noise, sensitivity to physical pain.

To proactively respond to the person's needs, in partnership.

Patient not to be stressed or rushed.

Implementation :

The nurse followed the intended plan however there was a complication which needed creativity, adaptability, patience and calm especially, on the part of the nurse.

Complication requiring immediate response :

Patient began to spasm severely immediately she attempted to eat. Food fell everywhere, patient became distressed, tearful, breathing difficulties and swallowing difficulties manifested.

Response :

maintain valuing posture . Wait for spasms to subside. Maintain silence but thinking what to do to help the patient. Patient tried several times to eat unsuccessfully, reduced ability to bite rice

A Severe ME-aware nursing model (*continued*)

cake Patient suggested breaking the food into small pieces and placing directly in mouth. Replaced lost food. Successful intervention.

Evaluation :

Patient evaluation : Was surprised by the strength of spasms and shocked by the loss of ability to even bite a rice cake. Grateful for the calm and valuing posture and support in being enabled to eat, because she needed the food to avoid hypoglycaemia. Shaken by the intensity of the experience.

Nurse's evaluation :

Mind : Pleased they had the knowledge and insight to appreciate the complexity of symptoms .

Body : Remained very still and maintained an open, calm and affirming posture.

Emotion : Felt concerned and dismayed yet managed to convey warmth and positive unconditional valuing.

Spirit : Maintained stillness and centeredness, despite distressing circumstances and maintained partnership stance, open for patient's communication.

Learning : although meal planned well, with awareness of potential symptom issues, the strength of the spasms and the rapid deterioration was unexpected in reality. Even under stress and with patient distress the nurse responded as intended. Replacing the food was an important aspect.

Conclusion

New ways of enabling nurses to assist patients with ME/CFS urgently need developing . The starting point, as this article has stressed , must be awareness that ME/CFS is a neurological disease , with multi-system dysfunction. Some of the complex environmental hurdles that ME/CFS sufferers have to overcome in order to access care have been detailed.

A self –reflective, partnership-based model of practice has been outlined, in order to begin to meet the complex needs of these still neglected patients.

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The Terminology of ME & CFS

By Professor Malcolm Hooper

The term BENIGN MYALGIC ENCEPHALOMYELITIS was first introduced in the UK in 1956 by a former Chief Medical Officer (Sir Donald Acheson) and not by Dr Melvin Ramsay as is sometimes claimed.

The word "benign" was used because it was thought at the time that the disorder was not fatal (as poliomyelitis could be, with which it had some similarity), but it was quickly realised by clinicians that ME was not a "benign" condition, as it has such high morbidity (i.e. such a lot of suffering and ill-health), so by 1988 clinicians had stopped using the word "benign" and referred to it as ME, the first to do so being Dr Ramsay.

However, the ICD still uses the term "benign" in its classification.

MYO relates to muscle
MYOSITIS = inflammation of muscle

MYALGIA = pain in muscles (pain that is called "myalgic")

MYOPATHY = any disease or disorder of muscle

MYEL (or MYELO) relates to the spinal cord (the main nerve in the body)

MYELITIS = inflammation of the spinal cord (NB. Not to be confused with the other meaning of myelitis, which = inflammation of the bone marrow, as in osteomyelitis)

MYELIN SHEATH = a layer of fatty white material that surrounds and insulates nerve fibres

DEMYELINATION = the loss of this protective insulation round nerve fibres (as seen in multiple sclerosis and sometimes also in ME)

ENCEPHALON = the brain

ENCEPHALO = relating to the brain

"ITIS" on the end of a word = inflammation (e.g. hepatitis = inflammation of the liver)

So, ENCEPHALOMYELITIS = inflammation of the brain and spinal cord

BENIGN MYALGIC ENCEPHALOMYELITIS therefore means a non-fatal disorder (inflammation) of the brain and spinal cord, with pain in the muscles

ENCEPHALOPATHY = any non-inflammatory disorder affecting the brain

Despite the claims of some psychiatrists, **IT IS NOT TRUE THAT THERE IS NO EVIDENCE OF INFLAMMATION OF THE BRAIN AND SPINAL CORD IN ME**: there is, but these psychiatrists ignore or deny that evidence. For example:

1988 In conjunction with the University of Pittsburgh, the US NIAID held a large research workshop called "Consideration of the Design Studies of Chronic Fatigue Syndrome".

There were participants from the Centres for Disease Control and from the National Institutes of Health.

One of the presentations was by Dr Sandra Daugherty, who reported that MRI scans on patients demonstrated abnormalities consistent with demyelination and cerebral oedema in 57% of patients studied. (It was at this conference that it was recommended that the term "CFIDS" be used instead of the term "CFS" on the basis of the immune dysfunction that had been observed in the disorder).

1989 Detection of Viral-Related Sequences in CFS Patients using Polymerase Chain Reaction W.John Martin (Nightingale Research Foundation: 1989: 1-5

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The Terminology of ME & CFS

By Professor Malcolm Hooper

1990 Chronic Fatigue Syndrome and the Psychiatrist SE Abbey, PE Garfinkel Canadian Journal of Psychiatry 1990:35:7:625-626

1992 A Chronic Illness Characterised by Fatigue, Neurologic and Immunologic Disorders, and Active Human Herpesvirus Type 6 Infection D Buchwald, PR Cheney, R Gallo, AL Komaroff et al Annals of Internal Medicine 992:116:2:103 This paper states "Magnetic resonance scans of the brain showed punctate, subcortical areas of high signal intensity consistent with oedema or demyelination in 78% of patients"

1994 Detection of Intracranial Abnormalities in Patients with Chronic Fatigue Syndrome: Comparison of MR Imaging and SPECT. RB Schawrtz, BM Garada American Journal of Roentgenology 1994:162:935-941

1995 Pathophysiology of a Central Cause of Post-Polio Fatigue Richard Bruno et al Annals of the New York Academy of Sciences 1995:753:257-275

1997 A 56-year old woman with chronic fatigue syndrome Anthony J Komaroff JAMA 1997:278:14:1179-1184

It is true that there is no evidence of inflammation of the brain or spinal cord in states of chronic fatigue or "tiredness."

It is also true that neither the 1991 (Oxford) criteria nor the 1994 (CDC) criteria select those with ME, as they both expressly include those with somatisation disorders and they expressly exclude those with any physical signs of disease (as is the case in ME), so by definition, patients with signs of neurological disease have been excluded from study.

It is also true that Professor Simon Wessely and his colleagues use the terms "fatigue", "chronic fatigue", "the chronic fatigue syndrome (CFS)" and "myalgic encephalomyelitis (ME)" as synonymous. Such obfuscation has greatly hindered research, as pointed out in the 1994 Report of the National Task Force on Chronic Fatigue Syndrome (CFS), Post-Viral Fatigue Syndrome (PVFS) and Myalgic Encephalomyelitis (ME), published by Westcare, Bristol and supported by the UK Department of Health, which stated:

"Chronic fatigue syndromes remain poorly understood. Progress in understanding them is hampered by:

- the use by researchers of heterogeneous study groups
- the use of study groups which have been selected using different definitions of CFS
- the invalid comparisons of contradictory research findings stemming from the above".

The Report names psychiatrists Dr Simon Wessely, Dr Peter White and Dr Michael Sharpe and acknowledged their help, but then makes the point that "people who gave their help are not necessarily in agreement with the opinions expressed" (page 87). It was said to be because those psychiatrists strongly disagreed with the findings of the 1994 Westcare Report that in 1996 they produced their own report (the Report of the Joint Royal Colleges on CFS (CR54), which was internationally recognised as being biased and seriously flawed).

Classification

The WHO was founded in 1948.

The International Classification of Diseases (ICD) comes in two volumes: Volume I is the

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The Terminology of ME & CFS

By Professor Malcolm Hooper

Tabular List and is a list of codes plus the name of the condition which goes with that code. Volume II is the Code Index, which alphabetically lists all the phrases and names of conditions commonly used for a condition, together with the appropriate code.

The Tabular List (Volume I) does not list everything which is in the Code Index (Volume II).

Benign myalgic encephalomyelitis (ME) has been classified in the International Classification of Diseases (ICD) as a neurological disorder since 1969, when it was included in ICD-8 at Volume I: code 323: page 158 and in Volume II (the Code Index) on page 173. (ICD-8 was approved in 1965 and published in 1969).

Prior to 1969, the term benign myalgic encephalomyelitis (ME) did not appear in the ICD, but non-specific states of chronic fatigue were classified with neurasthenia under Mental and Behavioural Disorders.

Benign myalgic encephalomyelitis (ME) was included in ICD-9 (1975) and is listed in Volume II on page 182.

The term "Chronic Fatigue Syndrome" was not introduced by Holmes et al until 1988 and therefore did not appear in the ICD until 1992, when it was listed as an alternative term for benign myalgic encephalomyelitis (ME). Another alternative term listed is Post-Viral Fatigue Syndrome.

In ICD-10 (1992), benign myalgic encephalomyelitis (ME) continues to be listed under Disorders of the Nervous System at G93.3, with the term Syndrome, Fatigue, Chronic, as one of the descriptive terms for the disorder.
By contrast, in ICD-10 (1992), neurasthenia and other non-specific syndromes of on-going or chronic "fatigue" are listed at section F48.0 (Volume I, page 351).

Non-specific states of chronic fatigue are classified as Mental and Behavioural Disorders, subtitled "Other Neurotic Disorders". Note: benign myalgic encephalomyelitis (ME/CFS/PVFS) is expressly excluded by the WHO from this section.

Note also that the WHO has confirmed in writing that -

"it is not permitted for the same condition to be classified to more than one rubric as this would mean that the individual categories and subcategories were no longer mutually exclusive".

Therefore, ME/CFS cannot be known as or included with neurasthenia or with any mental or behavioural disorder.

Professor Malcolm Hooper

From
http://www.investinme.org/Article%20010-Encephalopathy.htm

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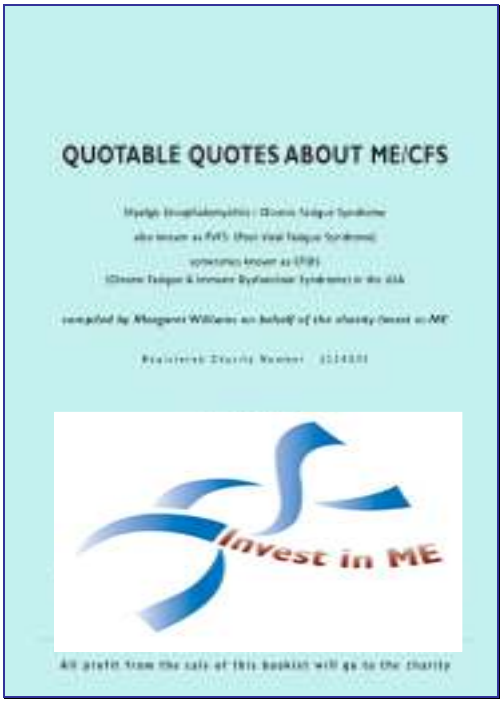

Quotable Quotes on ME/CFS

This 42 page booklet has been compiled by Margaret Williams and contains a plethora of quotes from ME experts and from others relating to ME, ME/CFS, CFS/ME and CFS. This is an invaluable document for researchers, healthcare staff, politicians, media, ME support groups and people with ME.

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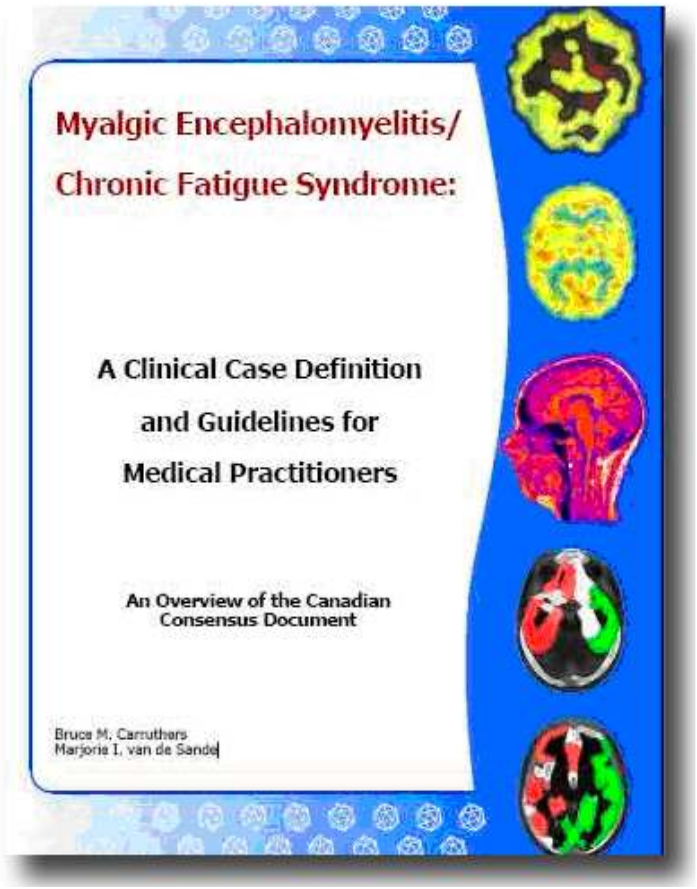

Invest in ME are the UK distributors for the Canadian Guidelines.

Described even by NICE as "*the most stringent*" guidelines available these are proper, up-to-date clinical guidelines which can also be used as a base for research criteria.

Findings from the study by Leonard A. Jason PhD (Comparing the Fukuda et al. Criteria and the Canadian Case Definition for Chronic Fatigue Syndrome) indicated that the Canadian criteria captured many of the cardiopulmonary and neurological abnormalities, which were not currently assessed by the Fukuda criteria.

The Canadian criteria also selected cases with 'less psychiatric co-morbidity, more physical functional impairment, and more fatigue/weakness, neuropsychiatric, and neurological symptoms' and individuals selected by these criteria were significantly different from psychiatric controls with CFS.

The Canadian Guidelines provide a means for clearly diagnosing ME and were developed specifically for that purpose.

They are an internationally accepted set of guidelines for which many in the ME community has been campaigning to be adopted as the standard set of guidelines for diagnosing ME.

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Wheelchair Use and Attitudes

By Sue Pearkes

Sue Pearkes has had Myalgic Encephalomyelitis since January 2007 and has been using a wheelchair for about a year.

There is a message which needs to be publicised about wheelchairs, to the three groups of people involved: the medical professions, the disabled community, and the able-bodied population at large.

Emotional Overtones

For some extraordinary reason (historical, perhaps?) there is an emotional subtext attached to wheelchairs. The able-bodied population tend to avoid or ignore them, possibly motivated by fear that they might "catch" disability; wheelchairs make the able-bodied uncomfortable, and they fear the possibility of ending up "in-a-wheelchair" (all one word).

There is also the feeling that if one starts using a wheelchair, one has "given up"—one should fight to the utmost to keep out of the wheelchair, regardless of the pain, discomfort and curtailing of activities that one experiences as a result.

The disabled community is also affected by this attitude—those who have been able-bodied will tend to have the same fears as able-bodied people, and those who have always been disabled will be influenced subconsciously by the existing negative attitudes of the able-bodied population. There is also the feeling that if one starts using a wheelchair, one has "given up"—one should fight to the utmost to keep out of the wheelchair, regardless of the pain, discomfort and curtailing of activities that one experiences as a result. (I consider "giving up" and "acceptance" to be two totally different concepts, incidentally.) I have a friend with cerebral palsy who, with advancing years, started to suffer badly with arthritis, and when he finally "gave in" and started using a wheelchair, found that it transformed his life, and he wished he had started using it years ago.

These attitudes are not helped by the medical professionals who, being human beings, will also often be influenced by these negative attitudes, having lived in the able-bodied community all their lives, before, during and after qualifying in their professions (I am including nurses, physiotherapists, occupational therapists etc. in "medical professions"). As professionals, they might be expected to help to improve the situation, but in actual fact they perpetuate and reinforce it. They may be conditioned to see wheelchairs as a symbol of failure to achieve "healing" in their patients, and would therefore be very reluctant to promote something which they subconsciously believe shows them in a negative light. While they may need to warn people of the dangers of over-use of a wheelchair (muscle atrophy etc.), they should credit their patients with enough commonsense to use the wheelchair in a responsible way. They should ensure the right balance between general health and increased mobility; not indulging in a blanket rejection of wheelchairs, but considering the

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needs and circumstances of the individual patient, and in particular, listening to the patient's views and desires. The patient, after all, is the one who best knows his or her body and circumstances, and is living with the disability on a daily basis. Diminishing someone's ability to get around, or even to leave their house, or to condemn them to a daily grind of pain, by preventing them from using a wheelchair, can have an adverse effect on health; just being able to move around more easily, and to get out and about and socialise, surely has great health benefits.

A Mobility Aid

My attitude towards wheelchairs is that they are no different from glasses. You wear glasses to see better, and to improve your quality of life. You use a wheelchair to get around more easily, and to improve your quality of life.

The Wheelchair User

Most people (from all three groups, probably, but especially the able-bodied population) have no concept of the part-time wheelchair user. Most people think you are "in-a-wheelchair" (all one word) because you "can't walk," and if you *can* walk, you don't need a wheelchair. I know I cause people a lot of confusion when I get out of my wheelchair and pull it up steps into shops etc. People often think that if you move your legs, or get out, then you are a fraud and don't need the wheelchair. I have even been challenged by total strangers over this, as if it's any of their business.

"In-a-wheelchair," "wheelchair-bound," "confined-to-a-wheelchair," are all extremely emotive and negative phrases. Not thinking about this until I was disabled, I thought that the phrase "wheelchair user" was a bit of politically-correct-speak. Now, however, I always refer to myself as a "part-time wheelchair user" and realise how important it is to be accurate in this respect. People do not become super-glued to their wheelchairs, becoming a single, freakish entity in the process. This is reminiscent of when the Conquistadors first arrived in South America, and the resident population had never before seen a man on horseback. They assumed that the two together were one unit; some sort of bizarre new creature they had never seen before.

My own experience has been interesting. When I mentioned to my GP last year that I was intending to get a wheelchair (I got it privately so didn't have to humiliate myself by asking for one from a profession that is so against recommending them!!) she gave the knee-jerk response, "Oh. We don't like wheelchairs very much. People use them all the time and then their legs don't work any more." Professionals who say this should credit us with a little common sense. I took no notice of her, knowing full well that a wheelchair would help me, and this has proved to be the case. I got it in time for our holiday last year, and without it I could not have done any of the things the others did; as it was I participated fully, and even did some things the others did not! Since then, it has enabled me to get out and about and do things without causing me great physical discomfort and pain, or completely exhausting myself for the next few days.

The other professions involved in my care

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ME STORY

I was assessed for one ME clinic but they said I was too disabled and that there were other issues that needed to be worked on.

They also said that because I was confined to a wheelchair they thought that would be too upsetting for the other members of their group!

- Gary

Wheelchair Use and Attitudes

(continued)

were much more positive. When the occupational therapist came to assess me at home, the first thing she saw when she came in was the wheelchair, and she said, "Oh good, you've got a wheelchair already." From this I assumed that had I not got one, she would have recommended one for me. When I saw the physiotherapist at the hospital, she commended my healthy and balanced attitude not only towards my illness, but also towards the mobility equipment I have, saying, "You have got your stick, crutches, trolley and wheelchair, and you pick and choose what you want to use according to your need at any given moment." Neither of these two professionals expressed any

The standard wheelchair, with its steel frame, is heavy, unwieldy, old-fashioned and ugly. The design has remained unchanged for decades. No wonder many people wouldn't be seen dead using them, particularly young people, who tend to be style-conscious.

negative attitude towards the wheelchair, or towards me for using it.

When I saw my GP again recently, I expressed how much the wheelchair had improved my quality of life, and how the other professionals had approved it and encouraged me. I hope she was able to take this on board and realise that an out-of-hand rejection of wheelchairs is not useful or helpful, and that there is more to the picture than the danger of muscle atrophy.

My own approach to my wheelchair has, I hope, challenged the preconceived attitudes of those who know me, and those I meet when out and about. I have decided that if I am to use one, then I might as well make a statement with it, and have decorated it. I started at Christmas, with baubles, tinsel and lights, and got so much positive feedback that I was amazed and delighted. Total strangers would approach me, wreathed in smiles, and say how cool it was, and they would engage me in conversation, as a creative individual, and not as "someone-in-a-wheelchair" (all one word). When Christmas was over and I had to take the decorations off, suddenly I was invisible again. I couldn't believe the difference. I decided to do something about it and put on flowers and lights, and immediately I got the positive reactions again. I also have decorated spoke-guards; these are available already decorated, or one can purchase plain ones and decorate them oneself, as I have done. They are a great way to express one's personality and elevate the wheelchair from an anonymous, functional object to the status of fashion accessory.

Wheelchair Characteristics

Maybe one reason why many disabled people are reluctant to start using a wheelchair is that the wheelchairs themselves are so uninspiring. I am fortunate enough to be the owner of a modern, ultra-lightweight wheelchair with a low back, cambered wheels, optional push-handles and minimalist appearance. Recently, while it was away having some work done, I had to resort to a borrowed NHS-type wheelchair which convinced me even more about the need for decent wheelchairs to be made available to everyone.

The standard wheelchair, with its steel frame, is heavy, unwieldy, old-fashioned and ugly. The design has remained unchanged for decades. No wonder many people wouldn't be seen dead using them, particularly young people, who tend to be style-conscious. They have fixed axles, a fixed back angle, an uncomfortable seat even with a good cushion, cumbersome armrests and enormous footrests which would put the average ice-breaker to shame. Using this type of wheelchair for a week (even part-time) made my back ache and my arms extremely tired, and I found it hard to maintain a good posture. The position of the wheels,

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centre of gravity etc., combined with the weight, made it impossible to do even the smallest wheelie, leaving me feeling as though I were glued to the floor. The turning circle was much too large and it lacked real manoeuvrability. The back was too high, and combined with the large armrests, this left me with a feeling of being trapped in a steel box, virtually unable to move. Finally, the push-handles on this type of chair convey the negative message, "I am a helpless baby/cripple, push me!" Their very presence encourages well-meaning able-bodied people to push the user, whether they want it or not. Most disabled people prefer not to be pushed if possible, as they value their independence and autonomy as much as any able-bodied person, and have no desire to be moved around by anyone else, and at a speed not of their choice. Being pushed, especially by someone inexperienced, can be alarming, and make one feel vulnerable and out of control.

Modern ultra-lightweight wheelchairs are a totally different proposition. They were originally designed by disabled veterans returning from the Vietnam War, who were dissatisfied with the wheelchairs on offer, and re-engineered the wheelchair from the ground up. Their design features give rise to a radically different appearance. Having a rigid frame made of modern lightweight materials such as aluminium alloy or titanium, and doing away with the added bulk of a folding mechanism, large footplates, unnecessarily high back and handles, reduces the weight dramatically, which obviously benefits people with all kinds of disability, especially those with limited energy or muscle power. The low back, while giving excellent support to the lumbar region, allows for total freedom of movement for the upper body, and encourages good posture; I have often been asked whether I need more adequate support for my back, but I reply that on the contrary, the support is exactly where I need it. Having the axles mounted further forward (the position is adjustable, as are many other features of these chairs) improves the efficiency of each push on the wheels as the user does not have to reach so far behind in order to obtain an adequate range of rotation. Self-propelling with a standard chair, the high back gets in the way, and one cannot get an adequate push. Of course, having the axles mounted further forward places the centre of gravity further back and compromises the stability somewhat, but this is balanced by increased energy efficiency

Most disabled people prefer not to be pushed if possible, as they value their independence and autonomy as much as any able-bodied person, and have no desire to be moved around by anyone else, and at a speed not of their choice.

and manoeuvrability and control of the chair. Going up steep ramps certainly increases the risk of tipping over backwards, but I have learned by experience that this difficulty can be overcome by going up backwards. (By doing this, one is also using one's biceps to *pull* oneself up, rather than the weaker triceps to *push*. This is reminiscent of reverse gear in the car being a very low gear and giving extra power.) Anti-tip tubes may help some users. If one does require pushing in a modern lightweight, optional push-handles are available, which can be temporarily inserted into brackets and removed again; alternatively some wheelchairs have discreet handles which fold down out of sight when not in use.

Having one's centre of gravity virtually over the

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axles enables the user to do wheelies with ease; once trained in this technique, it is liberating. Even quite large obstacles, and uneven ground, and even gravel, can be negotiated by raising the front castors off the ground and moving on the two drive wheels only. When moving slowly on normal surfaces, one tends to use all four wheels, but at speed, or over uneven ground, the front castors need hardly touch the ground; this "cruise control" mode allows for less rolling resistance and greater efficiency on the part of the user, thus saving energy. Because of the design, and the lightness of the wheelchair, this does not require much upper body strength on the part of the user. All these features cause the wheelchair to become an extension of the user, and with practice and experience, movement can become easy and natural, and the environment can have a less disabling effect. The whole design, including the quick-release axles so that the wheels may be easily removed, makes it much easier for the disabled person to put the wheelchair in and out of the car, and to carry out other day to day activities independently.

Quite apart from all these design and engineering features which improve the use of the wheelchair, turning it into a hi-tech form of locomotion, the appearance in itself is of great benefit to the user. It is modern-looking, cool and sporty, and does not make the user look like an invalid. The absence of push-handles conveys the message that the user is independent and perfectly capable of managing without interference, which in itself improves one's sense of autonomy.

The Importance of Choice

It is not only the practical and functional aspect that is relevant, but the style element is also very important. People are out and about using their wheelchairs, and the appearance can have a profound effect on one's image; we express ourselves by our outward appearance and choice of clothes and hairstyle, and the appearance of one's mobility aids is of equal importance. NHS grey, clunky, heavy and old fashioned equipment, whether it be a wheelchair, stick or crutches, do nothing for a person who is style-conscious. For most wheelchair users, it is not a matter of choice; they need a wheelchair in order to function. The able-bodied population (the "shoe-bound") have a choice of design and style of the devices they use to interface with the ground, and would be justifiably outraged if some outside agency dictated from above what sort of shoes they should wear, regardless of their suitability or comfort. Why should wheelchairs, the devices many disabled people use to interface with the ground, be any different? Of course, the "does he take sugar" attitude prevails; the NHS remains largely paternalistic—"We know what is best for you"—because the poor little cripple cannot possibly think for himself, and if he expresses an opinion contrary to that of the professionals, he is deemed a "difficult patient."

The argument the NHS gives against prescribing these wheelchairs is cost. However, I have come across people who, in the days when they used NHS chairs, had to own more than one because they were always breaking, and they needed one in reserve to use while the other was being repaired. When used by full-time users, wheelchairs take a lot of punishment, particularly if the user enjoys an active lifestyle. The modern lightweights, however, are immensely strong, and their users generally have no problems with maintenance once they progress beyond the NHS standard. The NHS contract with wheelchair manufacturers must be enormous; if they were to start prescribing modern lightweights, the cost would come down, which would benefit everybody. It does seem wrong that in a society where the welfare state is supposed to provide wheelchairs for those who need them, people have to pay for wheelchairs that actually work for them.

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Wheelchair Use and Attitudes

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The fact that modern lightweight wheelchairs are much more adaptable and adjustable would also be a benefit; the "one-size-fits-all" NHS philosophy actually causes a lot of damage, discomfort and pain to users whose wheelchairs do not fit them. My week using the NHS Iron Maiden was enough to convince me of the truth of this. There is no way that I could maintain the lifestyle and independence I enjoy if my modern lightweight were to be exchanged for a standard NHS-issue chair.

The original modern lightweight wheelchairs were designed by wheelchair users. I consider this to be a crucial point. Able-bodied designers of wheelchairs do not have inside knowledge of what is needed in a wheelchair. If the NHS insists on using able-bodied designers and prescribers of wheelchairs, these people should at least be compelled to use them for a month or so, just to see what it is like, and experience for themselves what people really need and want.

The Need for Education

There seems to be a considerable need for education about wheelchairs and their use, amongst all three population groups. I do not know the best way to get this message across, but the purpose of this article is to reach as wide a forum as possible, where it can be read and acknowledged. In particular I should like it to be read by the professionals involved in the care and treatment of disabled people. It may make them stop and think about their attitude, at least—somehow we've got to get this message across. These professionals' entrenched attitudes are doing us more harm than good, and causing no end of distress, when their role and function in life should be to help us. Pushing people to expend their precious reserves of energy, when they could be helped by the sensible use of such a marvellous device as a wheelchair, is totally wrong. We need encouragement, sensible advice and affirmation, not obstruction and condemnation.

Finally, sitting down as opposed to standing up is not necessarily a negative thing. At the recent Beijing Olympics, the Aussies accused the Brits of earning most of their medals sitting down!!!

The Use of Aids for People with ME – an Alternative View

Let us contrast the previous article ***Wheelchair Use and Attitudes*** with the views expressed by NICE in their recent Guidelines for ME/CFS and the submissions from St Bartholomew's Hospital Chronic Fatigue Services to NICE in response to those same guidelines.

St Bartholomew's Hospital Chronic Fatigue Services is one of the CNCC clinics set up by the government for treating people with ME. As in the case of almost all of the CNCC clinics it offers a biopsychosocial view of ME and is headed by a psychiatrist.

These are the views submitted by St. Bartholomew's Hospital Chronic Fatigue Services for the NICE Draft Guidelines Development Group, in relation to such as wheel chairs for people with ME/CFS (source - http://listserv.nodak.edu/cgi-bin/wa.exe?A2=ind0709A&L=CO-CURE&P=R2063&I=-3&m=17215)

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(i) On Disability aids and equipment:

The NICE Draft text stated -

6.3.6.8 For adults and children with moderate or severe symptoms, provision of equipment and adaptations (for example, a wheelchair, blue badge or stairlift) to allow individuals to improve their independence and quality of life should be considered, if appropriate and as part of an overall management plan.

Comment from St Bartholomew's Hospital Chronic Fatigue Services:

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The Use of Aids for People with ME – an Alternative View

"We disagree with this recommendation.

Why should someone who is only moderately disabled require any such equipment? Where is the warning about dependence being encouraged and expectation of recovery being damaged by the message that is given in this intervention?

We are in no doubt that it is a powerful message for a therapist of any sort to provide such aids.

Our view is that such aids should only be considered by a multi-disciplinary therapeutic team as a whole, and usually in the context of providing a temporary means for a patient to increase their activity levels.

An example would be providing a wheelchair for a bed-bound patient as part of their active rehabilitation programme. In our opinion, such aids should never be seen as a permanent solution to disability in this illness."

Another part of the NICE Draft Guidelines:

1.3.1.8 For adults and children with moderate or severe symptoms, provision of equipment and adaptations (for example, a wheelchair, blue badge or stairlift) to allow individuals to improve their independence and quality of life should be considered, if appropriate and as part of an overall management plan.

In reply to this St Bartholomew's Hospital Chronic Fatigue Services wrote:

"Equipment and aids may hinder recovery as much as help it, and their prescription needs to consider both outcomes.

We believe disability aids can help a patient towards recovery if their use encourages a widening and increase in their own activities, on a temporary basis, as a means of supporting a rehabilitation programme. They should rarely if ever be used for patients with only moderate disabilities."

From their web site - [http://www.bartsandthelondon.org.uk/formedia/press/release.asp?id=1216] St Bartholomew's Hospital Chronic Fatigue Services claim that theirs is a centre offering pioneering treatment for CFS/ME.

"The centre is a unique partnership between three separate Trusts which allows patients to experience an integrated "mind and body" approach involving physicians, psychiatrists, psychologists, physiotherapists and occupational therapists."

The St Bartholomew's Hospital Chronic Fatigue service contains a recommended reading list on its web site which offers literature from well-known psychiatrists. The treatments offered at St Bartholomew's Hospital Chronic Fatigue Services are indicative of their approach to ME/CFS –

"The treatment options that are available at our service include Cognitive Behavioural Therapy (CBT) provided by Clinical Psychologists, Graded Exercise Therapy (GET) provided by Physiotherapists and a Return to Work Programme and activity management run by Occupational Therapy. "

and

"we are one of the study centres involved in the PACE trial. This large-scale trial is the first in the world to test and compare the effectiveness of four of the main treatments currently available for people suffering from CFS/ME. "

despite the PACE trials being condemned by ME patients for their use of the flawed Oxford diagnostic criteria [see http://www.investinme.org/Documents/Journals/Journal%20of%20IiME%20Vol%201%20Issue%202.pdf].

The Oxford (1991) criteria have been criticised for being too broad -- they specifically include those with psychiatric fatigue and they potentially capture people suffering from "fatigue" that occurs in 33 different disorders -- and for specifically excluding those with neurological disorders such as ME.

The Physiology of Exercise Intolerance in Patients with Myalgic Eencephalomyelitis (ME) and the Utility of Graded Exercise Therapy

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ABSTRACT

This review discusses the suitability of graded exercise therapy for the treatment of myalgic encephalomyelitis (ME), based on current knowledge of the underlying physiology of the condition and the physiological effects of exertion on ME patients. A large body of peer-reviewed scientific literature supports the hypothesis that with ME an initial over-exertion (a period of metabolic stress) in conjunction with viral infection depletes concentrations of the metabolic regulator glutathione, initiating a cascade of physiological dysfunction. The immune system and muscle metabolism (including the muscles of the cardiovascular system) continually compete for glutathione, inducing a state of constant stress that renders the condition chronic. The impairment of a range of functions means that subtly different suites of symptoms are apparent for different patients. Graded exercise therapy has proven useful for a minority of these, and the exacerbation of symptoms for the majority is not subjective but has a physiological basis. Blanket recommendation of graded exercise therapy is not prudent for such a heterogeneous group of patients, most of which are likely to respond negatively to physical activity.

Following exercise, patients with myalgic encephalomyelitis (ME) uniquely exhibit exacerbated symptoms and a suite of measurable physiological changes indicative of stress (sub-optimal metabolic performance; e.g. reduced respiration and heart rate, increased glycolysis and lactic acid production, and concomitant limitation of activity[1-5]). Although these symptoms may not be universal[6], a significant subgroup of ME patients are affected in this manner[7]. The issue of exercise is critical for the treatment of the condition as one school of thought recommends "graded exercise therapy" as a general remedy for ME whilst another recognises that exercise intolerance may have an underlying physiological cause that may actually be aggravated by physical exertion. This difference of opinion influences policy: graded exercise therapy is one of the principal recommendations of the current NICE draft guidelines for the treatment of patients "mildly to moderately affected" by ME (p. 21, lines 20 to 23) [8].

Although recent general reviews of ME exist[9-11], our aim is to specifically review evidence for the mechanisms by which physical activity affects ME patients, and to investigate how graded exercise therapy may help or hinder recovery.

Although no single randomised controlled study has yet attempted to investigate every aspect of ME, the combined weight of empirical evidence to date indicates that the condition is characterised by a complex series of events involving reserves of metabolic regulators such as glutathione, muscle metabolism and the cardiovascular system. A significant body of literature suggests that these imbalances are associated with a

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dysfunctional immune system impaired by viral infection. Indeed, a hallmark of ME is a range of symptoms, varying in extent between patients, suggesting that a range of functions are impaired to greater or lesser degrees.

ME typically follows a flu-like illness, with elevated concentrations of viral particles subsequently detectable in blood and muscle tissues[12]. Post-viral fatigue is a well established possible consequence of infection by a range of different viruses[13-17], with enteroviruses specifically implicated in the case of ME – elevated concentrations of viral RNA sequences resembling coxsachie virus B are detectable in muscle tissue[12]. Furthermore, the majority of the limited number of ME patients so far treated with antiviral drugs (interferons) were able to return to work following treatment[18], also suggestive of a persistent 'smoldering infection'[19].

Crucially, post-viral fatigue is not related to the muscle disuse and deconditioning that can result from the initial period of illness[12]. Indeed, the mechanism underpinning post-viral fatigue is a multifaceted physiological imbalance. Nijs and co-workers[20] found that, for ME patients, graded exercise resulted in faulty regulation of the immune system, specifically increased activity of the enzymes "elastase" and "RNase L". RNase L is a key component in the cell's virus detection system and is up-regulated in response to viral infection. However, elastase degrades RNase L and is normally involved in removing it from the cell when concentrations are too high. Why should both be highly expressed in ME patients? Elastase is activated and degrades the RNase L in the absence of metabolic regulators such as glutathione. (Glutathione is an amino acid complex that modifies enzyme activity throughout the body, and ME patients exhibit either lower concentrations or an imbalance between its active and inactive forms[21-23].) Thus the simultaneous over-activation and mis-regulation of this part of the immune system can be explained by glutathione depletion. A range of factors contribute to glutathione depletion in the general population, including infection, the oxidative stress induced by strenuous or sustained exercise, and the long-term elevation of the stress hormones cortisol and adrenalin[24]. Furthermore, glutathione is also involved in sustaining respiration (i.e. the production of chemical energy compounds such as ATP in the mitochondria) thereby providing energy for active tissues such as muscle. Thus muscle tissue effectively competes with the immune system for glutathione[25] – sustained physical activity reduces the amount of glutathione available to the immune system, resulting in immune dysfunction. Conversely, an overactive immune system reduces the amount of energy available for muscle tissue, also exacerbating oxidative stress, and can account for both the chronic fatigue and pain (by inducing lactic acid production) that characterise ME. Thus, following an initial period of stress, glutathione concentrations may be too low for the optimal function of both the immune system and muscle tissues, paving the way for both persistent viral infection and fatigue, both of which feedback from each other to render the condition chronic.

This situation is compounded by the fact that glutathione not only has a supporting role in the immune response but also directly inhibits the replication of enteroviruses by blocking the formation of one particular protein (glycoprotein B) shared by all – including coxsachie viruses. Indeed, glutathione concentration is a major factor influencing the expression of other persistent viral infections such as HIV[26-29]. Thus glutathione depletion not only suppresses the immune system, it leaves the body particularly defenceless against enteroviruses. Sustained exercise or stress can deplete glutathione

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concentrations to the point where viral RNA is no longer prevented from replicating, aiding either an initial infection or the renewed replication of previously blocked viral RNA present in muscle tissue and blood[27, 29]. Thus glutathione depletion is a strong candidate for 'the trigger for reactivation of endogenous latent viruses' in ME[30]. A small number of studies demonstrate that foods rich in glutathione or direct glutathione injection help to relieve fatigue in ME patients, and may clear active viral infections[31, 32].

Although the above studies have concentrated on skeletal muscle, the heart (and the postural leg muscle involved in pumping blood back to the heart) is not exempt from glutathione depletion. Thus the above mechanism can also account for the range of cardiovascular problems associated with ME, including orthostatic (standing) intolerance (reviewed by Spence and Stewart[33]). Patients with orthostatic intolerance 'have continuous disability and commonly have exercise intolerance'[33].

Together, this evidence suggests that chronic fatigue in ME is symptomatic of the following sequence of events: a period of infection or strenuous physical or mental activity results in glutathione depletion; this renders the immune system relatively ineffective, particularly against enterovirus infection; the immune system becomes constantly activated (and inefficiently governed) because it has insufficient resources (glutathione) to completely rid the body of viral particles; the constantly elevated energy demand of the immune system detracts from other metabolic functions (particularly energy-demanding systems such as skeletal muscles and the cardiovascular system); limitation of respiratory and cardiovascular systems further locks the patient into a vicious cycle of inefficient energy production and use; increased reliance on anaerobic metabolism leads to lactic acid production and associated muscle pain.

Clearly, the performance of energy-demanding activities such as exercise can only aggravate this situation. Indeed, 82 % of ME patients in a recent study stated that graded exercise therapy worsened their condition, and only 5 % found it useful (compared to 70 – 75 % of patients who found either pain management or 'pacing' of daily activities useful)[34]. Furthermore, the Canadian Clinical Treatment Protocol warns that "externally paced 'Graded Exercise Programs' or programs based on the premise that patients are misperceiving their activity limits or illness must be avoided"[35]. If exercise is so detrimental, why is graded exercise therapy often recommended as a treatment for ME? Firstly, many of the studies cited here are recent, and the information and implications have perhaps not yet filtered up to policy makers. Secondly, the reclassification of ME as an ambiguous 'chronic fatigue syndrome' (CFS) by members of the psychiatric profession assumes that the symptoms have no physiological basis and are best treated with the traditional psychiatric method of facing and overcoming a problem, rather than direct removal of the problem at source. However, this approach jumps from hypothesis to treatment without investigating the mechanisms involved, perhaps explaining why "no psychiatrist has ever cured an ME patient using psychiatric treatments"[19]. Psychiatry, by definition, should not have authority over the treatment of physiological disorders, particularly those that occur chiefly in muscle tissues. Graded exercise therapy is founded on, and perpetuates, the myth that ME patients are simply malingering, while most are frustrated by their incapacity to satisfactorily conduct critical aspects of daily life[34].

ME is a heterogeneous disorder that affects different patients to varying degrees and with

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subtly different suites of symptoms. At best, graded exercise therapy has relieved symptoms for (but not cured) a tiny minority of patients, whilst the weight of empirical evidence indicates that exercise has direct and persistently negative impacts on the physiology and quality of life of a significant subgroup of ME patients. Any universally applied therapy is unlikely to address the heterogeneity of ME, and graded exercise is particularly unsuitable as it may worsen the condition, and should not be generally recommended without a high degree of confidence that it will not be applied to susceptible patients: it is difficult to conceive of a more inappropriate therapy for ME. By increasing the risk of relapse and overall health risks, rather than reducing them, graded exercise therapy also risks increasing the burden of illness on society at large. The present review suggests that an approach based on treatment of the underlying physiological dysfunction will be more fruitful.

Abbreviations

ATP = Adenosine triphosphate, RNase L = 2',5'-oligoadenylate (2-5A) synthetase/Ribonuclease L

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From CBT, GET And Human Rights:

by R. Mitchell and V. Mitchell

from

http://www.investinme.org/Documents/PDFdocuments/CBT%20GET%20and%20Human%20Rights.doc

Contrast the intellectual and scientific rigour applied in the approval process for the licensing of drugs for clinical use, with the lack of scientific and intellectual rigour applied in the NICE draft with regard to the recommendations for the use of Psychological Therapy in CFS/ME. When compared with the extensive clinical trialling over many years and the independent scrutiny a drug therapy is subjected to, the small and heavily criticised evidence base used to justify the recommendation of CBT and GET for CFS/ME in the NICE draft is seen to be totally inadequate.

In respect of informed consent, it cannot arise. There simply cannot be informed consent since there are important ethical, safety and regulatory questions arising from these treatments, to be addressed.

Ethical and safety questions such as those raised in the MRC Neuroethics Report 2005 should be paramount. It is hard to envisage any Independent authority clearing a drug for Human testing or use without ethical and safety issues, like those surrounding Psychological Therapy, being resolved.

By ignoring these serious issues with regard to Psychological Therapy, we believe that, as drafted, the Guidelines violate the right of clinicians and patients to the highest, safest standards of Medical practice and care, amounting to a violation of their Human Rights.

This is a Human Rights issue. Without an answer to whether this type of therapy is 'acceptable to Society' and if it is, without an effective Regulatory framework governing its development and use, there is the serious risk that sick and vulnerable people everywhere will be vulnerable to exploitation and abuse at the hands of the vagaries of power, politics and prejudice.

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The NICE GUIDELINES FOR ME/CFS - REASONS FOR REJECTION

On the 22 August 2007 The National Institute of Health and Clinical Excellence (NICE) published guidelines for doctors, titled: Chronic Fatigue Syndrome / myalgic encephalomyelitis (or encephalopathy) - Diagnosis and management for CFS/ME in adults and children.

Eight of the biggest ME organisations and more in the UK are strongly critical of the NICE guidelines and have declared them 'unfit for purpose'. They demand a considerable rewrite of the guidelines. We think, as they do, that the NICE guidelines will make the situation even worse for ME patients than it is at present.

Some of the critical points include:

Cognitive behavioural therapy (CBT) and graded/graduated exercise therapy (GET) are recommended as first line treatments for mild or moderate CFS/ME, and 'Activity Management strategy', which has elements of CBT and GET, for the most severely ill. These therapies have shown to have little effect (CBT) or are potentially harmful (GET). Large scale patient surveys in the UK show opposite results to the NICE guidelines. Apart from the outlined concerns, the key psychiatrists themselves, who actively promote these approaches say that CBT and GET cannot be described as 'curative' and/or have only a short term effect. (Michael Sharp, AACFS, (now IACFS/ME)) International CFS Conference, Cambridge, Mass., 10.-11. oktober 1998. S. Wessely, Editorial, JAMA 19.9.2001:286:11), Marcus JH Huibers + S. Wessely, Psychological Medicine, 2006:36(7):895-900).

CBT and GET are not specific methods for ME/CFS because the cause is unknown. Many have been made worse by these therapies. (Devanur & Kerr 2006): http://www.cfids-cab.org/rc/Devanur.pdf

The NICE Guidelines did not want to include or concentrate on research which actually documents the claims of the users.

Results of cognitive behavioural therapy and graded excercise therapy from large scale patient surveys

* 3074 patients (Jones, 2003)
  - CBT made no difference 55 %
  - CBT made worse 22 %
  - GET made no difference 16 %

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Norway's ME Association

The Norwegian ME Association, Norges Myalgisk Encefalopati Forening, was founded in 1987.

It has established self-help groups in many counties, and once a year all the group leaders gather in Oslo for an 'update' seminar, and to share their experiences and get new inspiration. Its office is in Oslo, centrally located behind the university. It provides factual information about ME to lay and health professionals, and helps and supports people with ME and their families and carers. Twice a year, it publishes a newsletter, and a magazine "ME-News" with medical articles and useful information. It works both nationally towards health authorities and government, and internationally to raise awareness of the seriousness of ME. The association is also a member of the Norwegian Federation of Organisations of Disabled People (FFO).

www.me-forening.no

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The NICE GUIDELINES FOR ME/CFS - REASONS FOR REJECTION

- GET made worse 48 %
- Pacing activity with rest was the most helpful 90 %
- Bed rest the most helpful 89 %

* 2338 patients (Action for M.E., 2001)
  - CBT helpful 7 %
  - CBT not helpful 67 %
  - CBT made worse 50 %
  - Activity managment most favourable 89 %
  - Rest most favourable 91 %

* 437 patients (25 % M.E. Group, 2004)
  - CBT helpful 7 %
  - CBT not helpful 93 %
  - GET helpful 5 %
  - GET not helpful 95 %
  - Psychotherapy helpful 10 %
  - Psychotherapy not helpful 90 %
  - The most helpful was activity managment and symptom control respct. 70 % - 75 %

At a conference in Fort Lauderdale, January 2007, Professor Fred Friedberg talked about a two year study in which patients used an actigraph (pedometer) to register their activity level. The patients reported subjectively increased activity levels, but at the same time the actigraph showed that the number of steps taken sank drastically. The results showed that graded excercise therapy did not lead to improvement in relation to increased total activity level (Friedberg 2002).

Physical activity exceeding "limit/ceiling effect" leads to increased symptoms and deterioration of the condition (Black & McCully 2005): http://www.dynamic-med.com/content/pdf/1476-5918-4-10.pdf

Patients can develop training intolerance, and this is shown by reduced activity level after 4-10 days. The inability to maintain an activity level, caused by worsening of symptoms, suggests that patients have reached an activity threshold. See also a more recent study by Yoshiuchi et. al (2007) which documents increased symptoms following graded excercise http://www.cfids-cab.org/rc/Yoshiuchi.pdf

It has been shown that patients with ME have increased oxidative stress during excercise, and this increase continues even after the the excercise has been stopped(Kennedy et al. 2005): http://www.cfids-cab.org/rc/Kennedy.pdf

It is important to note that patients do not protest about treatments which make them better, but they do protest against treatments which either do not work or make them worse.

Action for M.E. M.E. in the UK. Severely neglected. Membership survey, 2001.

http://www.afme.org.uk/res/img/resources/Severely%20Neglected.pdf

Jones DM. Some facts and figures on CBT, GET and other approaches, 2003.

http://www.meactionuk.org.uk/SOME_FACTS_AND_FIGURES_ON_CBT.htm

25 % M.E. Group. Severely Affected ME (Myalgic Encephalomyelitis) Report on Questionnaire, Issued January 2004.

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The NICE GUIDELINES FOR ME/CFS - REASONS FOR REJECTION

http://www.25megroup.org/Group%20Leaflets/Group%20reports/March%202004%20Severe%20ME%20Analysis%20Report.doc

VanNess JM, Snell CR. Stevens SR, Bateman L, Keller BA. FACSM. Using Serial Cardiopulmonary Exercise Tests to Support a Diagnosis of Chronic Fatigue Syndrome. Medicine & Science in Sports & Exercise: Volume 38(5) Supplement May 2006 p S85

http://www.acsm-msse.org/pt/re/msse/fulltext.00005768-200605001-01259.htm;jsessionid=HV9HJwhtvwtNlGyB7vvTzBpDQt0xbKl87pnqpqrSnCTT9QlWCNXx!65375592!181195628!8091!-1?nav=search&fullimage=true

VanNess and his coworkers (2006) have written the following:

"Reduced functional capacity and post-exertional malaise following physical activity are hallmark symptoms of Chronic Fatigue Syndrome (CFS). That these symptoms are often delayed may explain the equivocal results for clinical cardiopulmonary exercise testing (GXT) with CFS patients. The reproducibility of VO max in healthy subjects is well documented. This may not be the case with CFS due to delayed recovery symptoms. Conclusion: In the absence of a second exercise test, the lack of any significant differences for the first test would appear to suggest no functional impairment in CFS patients. However, the results from the second test indicate the presence of a CFS related post-exertional malaise. It might be concluded then that a single exercise test is insufficient to demonstrate functional impairment in CFS patients. A second test may be necessary to document the atypical recovery response and protracted malaise unique to CFS."

Both the Association of British Neurologists and The British Psychological Society have criticised the NICE guidelines.

Much of the research referred to, has been done on patients with fatigue, but who do not have ME.

The project has not been a cooperation where professionals and carers have taken part as it is described in the NICE guidelines. It is written that there was cooperation but the ones who have been involved as user representatives feel it was not real cooperation, but a form of masquerade. Considerable and documented contributions from users and experts who support the physical/organic cause of the illness have been ignored in a great degree. This and lack of real user contribution has also been confirmed in personal communication between organisations and Norway's ME Association. The documents have been delivered to Competence Network co/Cecilie Daae.

The NICE guideline's definition of the illness is so wide that it includes almost everyone with unexplained fatigue, and not ME, diagnostic code G93.3. There is a clear need to subgroup patients who fall under the umbrella term CF (fatique syndromes). The use of overview articles as research methods, have clear weaknesses when studies of heterogeneous populations are included - the methodology critique doesn't focus on the fact that the evidence base is very weak and the dropout analysis cannot find out who, and why, the dropout rate in a few studies is very high. See also the critique the Association has produced in relation to the Knowledge centre's report.

A lot of information on the physiological abnormalities was presented, but NICE has ignored this for the benefit of the "biopsychosocial model" of the illness. This is a clear action which favours political strategies instead of medical and scientific evidence. The urgent need for biomedical research to uncover the underlying cause(s) have not been taken onboard. NICE has failed on several "Key Items" (Key Items 3, 5, 8, 10 and

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20) in the use of AGREE INSTRUMENT (Appraisal of Guidelines Research and Evaluation, the AGREE Collaboration, Sept. 2001).

Costing report

The cost of these treatment strategies is based on assumptions, which the costing report says in the introduction. In addition the report comments on excisting uncertainties in the diagnosis and treatment of patients with CFS/ME. The high costs are expected to become even higher than estimated. The British organisations are questioning these costs for interventions which don't have documented effect, and which the ptients themselves don't want, at the same time when official bodies don't prioritise biomedical research.

Legal evaluation

The critique of the NICE guidelines for ME/CFS was taken to High Court in the Royal Courts of Justice on June 17 2008. Two named persons act as litigants. The judge concluded that there are grounds for a full hearing. It is estimated that the hearing will take at least two days. The date has not determined, but expected soon. (Editor: the date is set for 11 and 12 February 2009)

There is diminishing trust in NICE within the British population because its decisions are constantly criticised and challenged. One questions its evaluation process and whether some distinct groups are disadvantaged by the process. There is a separate report where a wide range of patient organisations, among others cancer and multiple sclerosis organisations, Alzheimer's Society and many other organisations for neurological and autoimmune conditions, have come forward with searing critique of NICE's conduct and evaluations process. (House of Commons Health Committee: National Institute for Health and Clinical Excellence (NICE). Written evidence. HC 503-II. 17. May 2007).

Facts on ME

Thyroid malignancy in ME/CFS patients greatly exceeds the normal incidence of thyroid malignancy in any known subgroup. The thyroid malignancy incidence in the ME/CFS group may exceed 6,000 / 100,000. As part of their investigation, Myalgic Encephalomyelitis / Chronic Fatigue Syndrome (ME/CFS) patients should be examined by thyroid ultrasound for evidence of thyroid pathology and malignancy. Thyroid pathology may be missed in this group of patients if investigation relies only upon serum testing for TSH, FT3, FT4, microsomal and thyroglobulin antibodies, which are usually normal. Thyroid uptake scans tend also to be normal and may also miss malignant lesions. A newly recognized syndrome may exist in ME/CFS patients characterized by: (a) thyroid malignancy, (b) persistent abnormal cortical and subcortical SPECT brain scans (NeuroSPECT), (c) failure of thyroidectomy surgery and hormone replacement to correct the fatigue syndrome, and (d) an unusual high incidence of cervical vertebrae osteoarthritic changes. ME/CFS patients with treated non-malignant thyroid disease and abnormal NeuroSPECT scans may also fail to improve despite adequate thyroid hormone replacement.

From Thyroid Malignancy Association with Cortical & Subcortical Brain SPECT Changes In Patients Presenting with a Myalgic Encephalomyelitis / Chronic Fatigue Syndrome. AJ38-2

Hyde MD, Byron
Leveille MD Jean
Vaudrey, Sheila
Green, Tracy

FROM 2 SCORE AND 5 TO 3 SCORE AND 10

A personal view of ME

by Nan Socolow

My story is similar to all other PWC's (*people with CFIDS = people with ME*) stories. First the signs and symptoms - bizarre, strange - unlike any other illness we've had. Then, the disbelief, the almost endless search for understanding physicians to name this disease, the expense of medical tests, treatments, forays into alternative therapies (is it all in my head? The mind/body connection?), the vials of useless medications and antibiotics adding injury and insult to our bodies and psyches. Finally, diagnosis and the shock that something chronic was wrong with us, the acceptance of a disease with a name even if the name was "Yuppy Flu" or "Major Kvetch Illness" or "ME" - Myalgic Encephalomyelitis. In short, CFIDS. And resoundingly, the resulting conviction that we are the canaries in the coal mine, the thin edge of the wedge of pollution of the water we drink, the food we eat, the air we breathe, pollution that is causing illness on Earth.

My CFIDS started in 1983 when Epstein Barr and Post Viral Fatigue Syndrome were the buzzwords. I was a vital 45 years old, divorced, active and hardworking mother of 3 teenagers, maybe a type A alpha female in linen jackets, silk blouses, heels and hose and smartly bobbed hair. A flu, followed by some sort of existential, clearly felt defining moment - a "click" in my body - changed everything in my life from major to minor and I endured a draconian fluish feeling for months and months and months. The "hit by a truck" poleaxed feeling, the extreme hangover that never goes away even though alcohol is not tolerated, not even a sip.

You are familiar with the symptoms - if you're reading this piece in the Chronicle - I won't list them here. But if you've almost fainted in detergent and soap and scented candles and air "freshener" aisles of your supermarket, almost keeled over upon entering a department store with the scent of tung oil on new clothing, almost collapsed upon having your car's gas tank topped off, almost passed out from the sensory input (aural, physical, emotional, etc) in any airport or crowded public place, reeled from a few sips of beer or champagne or a gin and tonic, then you've been there, too. The remissions and flare-ups. The awful days, when one could barely get out of bed, and the better ones when a drive to the market was a possible endeavor. The little wee walnut-sized life when everyone else is out there in the can-do life broad as Montana.

The good news is that I have had CFIDS for the past 25 years. The bad news is that I have had CFIDS for the past 25 years. I am now 70 years of age, to my great

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Nan Socolow

Nan Socolow is a poet who has lived for 20 years on a small island 90 miles south of Cuba in the British West Indies. She was Director of Development at Ford's Theatre in Washington, DC, Administrator of Rockefeller College at Princeton University, a Language Services Escort Officer of the US State Department and United States Information Agency. She worked in the White House during President Carter's Administration on the first Arab-Israeli Peace Treaty Signing events. She has three children and four grandsons and CFIDS).

FROM 2 SCORE AND 5 TO 3 SCORE AND 10

surprise, and looking forward to 2 plus decades, perhaps. In our case - in the case of we who contend with and accept the constraints and miseries CFIDS imposes, financial, emotional, physical - contrary to what Nietzsche said, whatever doesn't kill us lets us keep on living with CFIDS.

Instead of moaning or grieving or thinking of what is lost and past, I am grateful for what remains. For the remains of the day. A small home with no stairs, children and grandchildren in touch though living in far-away countries (Singapore, Russia), friends constantly "there" on the internet and through email and in person. I still enjoy reading, writing, gardening, cooking, intellectual pastimes (i.e. thinking about sex but not doing much about it, raging and ranting about politics and voting by email, helping those less fortunate, playing scrabulous and earning freerice.com online and laughing hard as often as possible) and the very rare social occasion. A lunch with friends. A dinner out. A warm bath in the sea. Renunciation of consumerism. I used to wear glad rags, evening clothes, bikinis and the like. Now I wear Saucony sneakers in different colors and long pants, skirts, tshirts, shorts, and am fully dressed with a smile. Comfy. And make-up - what the heck - mascara, lipstick, blush to put as pleasant a phiz as possible on the face of this illness. And I watch my every footstep and try to avoid major hassles, aggro and agita.

After burning out in two very high stress jobs in my 40s, in the 1980s (working for an Ivy League University as Administrator of a College of 500 freshmen and sophomores all brilliantly fraught, and then working for the US State Dept and USIA in Washington DC accompanying guests of the US Gov't on all-expenses-paid 30 day trips around the US (sometimes 9 cities in 30 days with no downtime, on call 24/7 very best job and worst job I ever had), I found (with Canadian friends) a small British island 90 miles south of Cuba where I could live and work in a manana atmosphere in a hotel or real estate firm. A backwater, an island time forgot; only one hour by jet from Miami. A funny and friendly and laidback place; sign in a local's calabaza plot (calabaza, Caribbean orange pumpkin w. green skin, delicious) - "Don't Molest My Vegetables!". Cows still mosey down the island's one road with white cattle heron perched on their shoulders. The sea turquoise and calm and clear as glass. When asked "how are you" the locals reply "not as good as you", "can't complain", "keeping on keeping on" and "fine as sifted flour!".

The weather is wonderful here, hot and sunny and salubrious - always summer - most of the time, except during hurricane season when I lost my home in Ivan 4 years ago. Sea went right through my home like Grant through Richmond. Sherman through Atlanta. Katrina through the 9th Ward. I had insurance and so was lucky enough to rebuild - "all new stuff" - , but no longer have any attachment to "things".

So, there it is. A chronicle of decades of illness, but of hope as well. Hope that research and development will find a remedy for this global illness. Hope that the medical establishment will recognize this disease for the scourge it is, and will rename it something more worthy of respect like MS or ALS or AIDS or PD... instead of AST (Always Sick and Tired) or PCK (Pitiful Chronic Kvetch) or CFIDS, which doesn't do justice to the miserably life-changing aspects of this illness. And to all of us patiently bearing the burden of this disease with as much humor as we can muster, I wish us comfort and happy times, a great measure of joy, surcease from suffering, looking ahead and knowing that life is full of surprises, many of them happy.

Reasons why ME Does Not Belong to the MUS Category…and So Forth

By Norwegian ME Association

Invest in ME have translated this article which was kindly provided by the Norwegian ME Association
www.me-forening.no

1. ME does not belong to the description of MUS

The classic presentation of ME is as an illness with its own diagnosis and diagnostic code, and as such, ME does not fulfill the criteria of the MUS category as "not fitting any known diagnosis". Contrast this with an invitation to a seminar in the Health Directorate 26. September 2008. Dr Wyller writes that the diagnosis of Chronic Fatigue Syndrome (CFS) is not in the WHO ICD publication (2008:8). This is incorrect information which has been pointed out earlier. CFS is in the index with a reference to the diagnostic code G93.3. As CFS refers to the same diagnostic code as ME, this means that the condition must be classified under the same code and not under a psychiatric illness (e.g. neurasthenia (chronic fatigue - is not the same as ME/CFS) with code F48.0)(ICD10, printed edition from 1992).

Many therefore refer to the description ME/CFS. See also KITH 2006. The illness and the illness presentation are not new, neither internationally or in Norway. According to infectious disease specialist and previously head of department at Ullevål University Hospital, Oddbjørn Brukbakk, the condition is described in classic, old medical literature in infectious diseases. The diagnosis myalgic encephalopathy/encephalomyelitis (ME)/Post-Viral fatigue syndrome does not belong to the umbrella term MUS for various reasons. This will be examined more closely below.

2. The WHO classification of ME/Post-Viral Fatigue Syndrome

The World Health Organisation (WHO) was established in 1948. Before 1965 the condition debility and undue fatigue in the international classification system was placed under code 790.1. The condition was not referred to as ME before 1965. So the first time the WHO referred to ME was in 1965 ICD-8. This was first officially published in 1969 9ICD-8: Vol I code 323, page 158; Vol II (Code Index) page 173). ICD-9 was approved in 1977, and ME was listed in the alphabetical index under code 323.9 in Volume II, page 182.

The World Health Organisation (WHO) approved ME/Post-Viral Fatigue Syndrome as an illness in its own right in 1969 (Marshall, Williams and Hooper, 2001), and the illness was given the following code in the international classification of diseases: ICD-10, 93.3 in the chapter of neurological disorders. According to the taxonomic system of the WHO's international classification system, it is not allowed to classify an illness in more than one category. The Norwegian healthcare officials have endorsed the classification system, something which legally binds the Norwegian doctors and healthcare officials into following

It cannot be in anyone's interest (clinicians, researchers, patients, healthcare officials) for doctors to classify an illness based on their personal understanding as to where an illness belongs.

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Reasons why ME Does Not Belong to the MUS Category
(*continued*)

this system. The system does not allow individual doctors for their own good to classify the condition as F48.0 under mental disorders as long as the criteria for ME are met. It is clearly mentioned under the diagnostic code ICD-10, F48,0 (neurasthenia/chronic fatigue/psychosomatic conditions) that this diagnosis cannot be given until Postviral Fatigue Syndrome/Benign Myalgic Encephalomyelitis (ME,93.3)(ICD-10,1999) has been ruled out. It cannot be in anyone's interest (clinicians, researchers, patients, healthcare officials) for doctors to classify an illness based on their personal understanding as to where an illness belongs. Such practice can lead to mistakes in investigation, diagnosis and treatment. In addition it will lead to incorrect information in the medical records, skews the prevalence numbers and leads to problems in comparing research studies etc.

3. The illness is approved as its own entity in other countries

The following information shows the illness is approved as its own entity in several countries.

Denmark

Now deceased, Professor Viggo Faber MD, knew the illness very well and states the following in one of his articles:

> *"… involvement of f.ex. ME/CFS among the somatoform is in contrast with many years of research in the USA and elsewhere in the western world, which has led to ME/CFS being acknowledged by the WHO …, and that one in USA and most of the European countries has noted it as a somatic illness giving entitlement to a pension…(there) are very stringent criteria for diagnosing ME/CFS."* (Faber, 2000:22).

Great Britain

In 1959 Dr Donald Acheson (later nominated Chief Medical Officer) published an extensive overview of ME entitled *The Clinical Syndrome Variously called Benign Myalgic Encephalomyelitis, Iceland Disease and Epidemic Neuromyasthenia*. In this overview ME is clearly seen as a **clinical entity**. The British Department of Health acknowledged ME as a **clinical, organic entity** in November 1989 (Hansard HoC: 27th November 1989: 353). Great Britain endorses the WHO ICD-10 and therefore has to follow this classification system. The diagnosis of ME was acknowledged as a **distinct clinical entity** by the Royal Society of Medicine in 1978 based on thorough work by Lyle and Chamberlain (1978) who had prepared an overview of epidemic neuromyasthenia (another description of ME) in the period 1934-1977. Here a citation of this by Emeritus Professor Malcolm Hooper (2007):

> *"In 1978 the Royal Society of Medicine accepted ME as a nosological organic entity. The current version of the International Classification of Diseases – ICD-10, lists myalgic encephalomyelitis under G93.3-neurological conditions. It cannot be emphasised too strongly that this recognition emerged from meticulous observation and examination."* (p. 466)
>
> *"Today, many patients with fatigue as a major feature of their illness – for example cancer, chronic obstructive pulmonary disease, depression – are being diagnosed with CFS. This has led to confusion, and has left clinicians, patients and carers without recourse to proper clinical and social support."* (p. 467)

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Reasons why ME Does Not Belong to the MUS Category
(*continued*)

Australia

The diagnosis was approved in Australia at the start of 1990.

USA

In USA the situation is different because they have compiled their own clinical version of ICD. The American CDC published a summary of Chronic Fatigue Syndrome and its Classification in the ICD 31. March 2001 by Donna Dean. It can be found in the archives of Co-Cure or at the following link: http://www.co-cure.org/ICD_code.pdf

In the summary it says that ICD-9 was published in 1975 and that the description Benign Myalgic Encephalomyelitis can be found in the alphabetical index and is referred to as code 323.9.

4. The illness was accepted and treated a long time ago in Norway – before 1990

ME is a syndrome diagnosis, and it has been documented that ME was accepted in Norwegian neurology from before 1990. In an article in *Tidsskrift for Den Norske Lægeforening* (1991;111(2):232) (Journal for The Norwegian Medical Association) a neurologist, chief consultant Ragnar Stien MD, employed by the Rikshospitalet in the neurology department, confirms that fatigue/tiredness is not a new condition. Dr Stien thought that the Fatigue Syndrome could partly have an organic cause. He thought that the most correct description to use was Post Viral Fatigue Syndrome, a diagnosis he himself had given to a number of patients. Dr Stien demanded that there was extreme asthenia, the patient had muscle pain during physical activity and evidence pointing to a viral infection before. He had examined 20-30 patients with this illness presentation in the 1980s. His impression was that the patients affected suffered from "abnormally strong fatigability" (p. 232). They had to rest "hours after minimal exertion". Even though at that time there was no scientific evidence to rely on, Dr Stien felt that the patients were so severely affected that the cause was organic.

Professor and specialist in general practice medicine, Dr Even Lærum, employed at the Institute of General Practice Medicine, Oslo, underlined the importance of performing a thorough physical examination. He had no objection in using the diagnosis of Chronic Fatigue Syndrome if the patient had extreme fatigue and one could not find other explanations. The treatment was symptom oriented, lifestyle changes and that patients should not put pressure on themselves (TNLF, 1991;111(2):232).The use of the diagnosis was also implemented at the same time by the Neurology department, Haukeland University Hospital. Dr Aarli and Dr Haukenes published an article on the illness in 1995. Here is an extract from this article:

> *"All experience so far has shown that this illness cannot be beaten by training, because enforced training seems to make the condition worse. This is similar to Post Polio Syndrome, where it has been shown that physical training often makes the muscular weakness worse. Acknowledgement by others that the symptoms are real can be important so as to avoid adding reactive extra symptoms." (Haukenes and Aarli, 1995:3021)*
>
> *"... it is patients who have had normal function and work capacity who after a viral illness present with considerable tiredness where causality seems to be connected to the infection as a triggering event "* (ibid.)
>
> *"It is well known that an acute infection can be followed by a fatigue syndrome that goes away. The special with this condition is that the fatigue, or exhaustion, lasts so long."* (p. 3017) "

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(*continued*)

"The clinical presentation...appears in immediate connection with an infection" (ibid.).

"Fatigue or exhaustion is the dominating symptom. Even light use of muscles brings on such a feeling of fatigue by the patient that he/she is unable to perform any type of work, often for several days. It is also characteristic that efforts and physical training worsens the fatigue. The physical fatigue has some similarities with myasthenia gravis and has led to the denomination neuromyasthenia." (ibid., p. 3018)

The same year Dr Harald J. Hamre published an article on ME which then was called Chronic Fatigue Syndrome. Here is reproduced some of what he wrote.

"After a thorough diagnostic clarification the patients need a stable, supportive primary care doctor contact, with intermittent diagnostic re evaluation. Support and adequate rest is crucial, based on experience. Many will be totally or partially unable to work for a long time. (Hamre, 1995:3043)

Patients with Chronic Fatigue Syndrome *"can have significant and long-term relapses if they are pressed for a too high level of activity, e.g. by declaring recovery prematurely ... They have a number of ... symptoms ... that the doctor should know and take seriously."* (ibid., p. 3044).

In 1995, Dr Kreyberg also published an article about Chronic Fatigue Syndrome. It can be read in its entirety on the Internet, and her review of the condition is not therefore referred to here:

http://www.med.uio.no/iasam/forepi/epidemiologi/me/artikler/Et_naergaaende_mote.pdf

5. Diagnosis and necessary investigations And So Forth

It is noted in the directorate's report (2007) that there are strict criteria for diagnosis. In the general practice medicine it is reported that a high proportion of patients present tiredness/fatigue. Extremely few of these get a confirmed diagnosis of ME (G93.3) after years of investigations. The general practice medicine has moreover their own coding system with various umbrella terms. The diagnostic code which is used most often in the general practice medicine is A04 (A, zero, four):

"The diagnosis is difficult because it cannot be confirmed by specific tests, laboratory tests or physical findings. The doctor has to build on the typical illness history and recognition of the clinical presentation. Fatigue is a non specific symptom in the line with fever and nausea and can be provoked by a number of factors. The aim for an operational definition must be a characterisation of this reaction so that it can be recognised clinically and can be limited against other conditions". (Social- and Health Directorate, 2007:7)

At the Ullevål University Hospital, Medical division, a diagnosis is given based on recognised criteria (Carruthers et al, 2003; Fukuda e al. 1994) and a specific diagnostic guide which was formulated by Dr Brubakk and Dr Baumgarten. Infectious disease specialist, previously head of department at

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the infectious disease department at Ullevål, Dr Brubakk, is very familiar with ME/Post Viral Fatigue Syndrome (PVFS) since as long as the 1980s.

The occurrence of ME can be compared to Multiple Sclerosis. This is also a diagnosis which demands special investigation. In Bømlo, Hordaland, there are 10 people registered with MS in the MS register. This is a municipality with 12.000 inhabitants. In the same area there are also 10 documented people with ME. In two of the families there is either ME or MS in first degree relatives. This points to clear genetic and immunological components.

At the Haukeland University Hospital, department of neurology, where there has been a "fatigue clinic" for 15 years, they say that disability has to be documented using validated scales such as Fatigue Severity Scale (Krupp et al, 1989), Fatigue Scale (Chalder et al, 1993) and SF-36 (Ware & Sherbourne, 1992). It is considered very important not only to measure physical fatigue, but also cognitive fatigue, because it is often the cognitive dysfunction that patients themselves find most disabling. SF-36 is a well known tool which includes different functional dimensions. Data from a ten year period show that people with ME have fatigue scores at the highest level, from about 23-30 (extreme values) when compared to fatigue in the population (Loge, Ekeberg, Kaasa, 1998). The ME group differs therefore clearly in having far higher scores for total fatigue than one finds in the Norwegian population. More about this can be found in the summary of the biomedical conference in Oslo in 2007 (Stormorken 2007):): http://www.me-forening.no/index.php?option=com_content&task=view&id=103&Itemid=2

Reeves and colleagues at the Centres for Disease Control and Prevention (CDC, Atlanta, Georgia, USA) have explained in a scientific article a clinical, empirical approach to diagnosing and defining CFS (Reeves et al, 2005). The study showed that patients who had been classified empirically as having ME/CFS, were significantly more disabled (measured using SF-36), more severely fatigued (measured by Multidimensional Fatigue Inventory) and had more frequent and more serious accompanying symptoms than patients with medically unexplained tiredness (MUS/MUPS). The study shows that the empirical definition (by including different fatigue scales) includes all aspects of ME/CFS

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which have been specified in the 1994 case definition, and identifies people with ME/CFS in a precise manner which can easily be reproduced both by researchers and clinicians. The empirical definition makes it possible to separate ME from depression and idiopathic fatigue. That said, Jason and Richman (2007) have criticised the empirical definition. The way Reeves and colleagues present it, it will lead to a clear broadening of the criteria in that the prevalence of ME/CFS will increase drastically, from about 800.000-1 million people to 4 million Americans. The critique against Reeves' empirical definition can be found at the following web address:

http://www.iacfsme.org/IssueswithCDCEmpiricalCaseDefinitionandPrev/tabid/105/Default.aspx

There is a reference to Reeves et al 2007 at:

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http://www.pophealthmetrics.com/content/5/1/5

Kathrine Erdman (2008) has published an article in which she explains the biomedical abnormalities that differentiate ME/CFS from depression: http://jaapa.com/issues/j20080301/pdfs/cfs0308.pdf Harvard-professor Anthony Komaroff has listed up to 10 central findings of biomedical abnormalities in ME/CFS: http://www.cfids.org/cfidslink/2007/062004.pdf Klimas and Koneru (2007) have written an overview of last year's advances in research. It provides a quick and easy introduction to different areas which document physiological disorders in ME and is highly recommended. ME is **not** unexplained, it has proven genetic factors, increased inflammation and many immunological changes. There are numerous findings, and one can no longer pretend that the biomedical research does not exist or look away from the biomedical factors in the illness presentation. Lorusso and colleagues (2008) come now up with an article which focuses on the immunological aspects in ME/CFS. They bring forward a high level of cytokines which can explain symptoms such as fatigue and flu like feeling and which can influence NK cell activity. The authors' hypothesis is that immunological factors form the basis for ME/CFS.

Who is best placed at giving the diagnosis?

Medicine is based a lot on clinical experience, such has it always been, but with so few patients per general practitioner, it will not be easy to build up enough experience. Based on feedback from patients the Association feels that at present general practitioners do not treat this group of patients in a good enough way (there are exceptions). If the diagnosis is given by a general practitioner, special training is necessary. At present with a demand for a specialist evaluation in NAV (Norwegian Labour and Welfare Organisation) regulations, extensive differential diagnosis and a lot of clinical experience, the Association can support a trial period of allowing general practitioners to diagnose because there is such a long waiting list for a specialist evaluation. The Association is worried that too many will be diagnosed because general practitioners lack adequate competence (see Dr Spickett's statements below). It is also pointed out in NAV's circular that *"The diagnosis of the condition is difficult and labour intensive, and ruling out normal tiredness and other illnesses can be difficult. It is therefore important to perform a thorough medical examination, especially to find out possible other illnesses that can be cured."*

http://rundskriv.nav.no/rtv/lpext.dll/rundskriv/r12/r12-01/r12-p12-06?f=templates&fn=document-frame.htm&2.0

Infectious disease specialist Dr Gavin Spickett (2008), specialist in immunology and lead clinician at the Royal Victoria Infirmary, Newcastle upon Tyne, stated at a ME/CFS conference in Cambridge (UK) 6. May 2008 that even though there were strict criteria for referrals to the CFS clinics, there were many who after further investigations turned out to have another diagnosis. ME is a very serious and rare condition. Because the condition is found only in 1-2 per 1000 people, a general practitioner might not have more than 1-2 people with this illness in their practice. Dr Spickett's presentation dealt with experiences with the so called CFS centres in Great Britain. His focus was on the key role of a medical examination of patients with suspected ME/CFS. When patients were referred to the centre, they underwent a thorough clinical evaluation to rule out other diagnoses that could explain the fatigue and to make sure that patients eventually could get correct treatment if there were other diagnoses. An overview of their work showed that experienced ME/CFS clinicians find other diagnoses among a large proportion of

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patients with referrals due to fatigue. The centre gets unnecessary large numbers of referrals of patients presenting with fatigue with questions about ME/CFS. This is despite the strict guidelines that were developed for referrals with specifications of the examinations that should be performed beforehand. Dr Spickett and his colleagues' experience shows that quite clear guidelines have not led to a reduction of patients who get another diagnosis in connection with investigations at the specialist centre. This is a clear indication of how difficult it is to diagnose and specialist competence is actually needed. This is especially important when there is no confirming diagnostic test and one depends on the use of internationally approved diagnostic criteria on every single patient. At present the general practitioners do not have enough knowledge of ME, some don't even believe in the diagnosis and many have big problems in dealing with this group of patients.

The Diagnosis is approved by the Social Security/ NAV – strict criteria

The State Social Security informed in a circular to local social services in Norway, 30. May 1995, that the condition must be accepted as an illness. The requirement was that certain criteria had to be fulfilled (Holmes criteria, 1988; Fukuda criteria, 1994). The State Social Security (now NAV) thought that this would involve a small amount of cases and these had to be evaluated in a wholly concrete manner. Dr Haukenes and Dr Aarli (1995) thought that the diagnosis of Post Viral Fatigue Syndrome (PVFS) should be used for this type of patients, but only after a thorough clinical evaluation. Therefore there were strict criteria for diagnosis. In their article Drs Haukenes and Aarli (1995) discussed the biomedical functional abnormalities that were known at that time.

The diagnosis was officially approved by the State Social Services in 1995 with the following description: G93.3. Post-Viral Fatigue Syndrome/ Benign Myalgic Encephalomyelitis (ME): Notification no 3/99. The illness must have brought on a considerable reduction in functional ability, i.e. more than 50 percent, where the revenue ability is reduced by more than half. The duration requirement is set to 3-4 years without sign of improvement in order to be awarded disability benefits. ME has been in the Norwegian version of ICD-10 given the diagnostic code G93.3. Before the diagnosis of ME can be given, MUPS (e.g. neurasthenia, chronic fatigue –F48.0) (ICD-10, 1991) must be ruled out. It is important to remember that both the NAV rules and regulations and the State Social Welfare law is legally binding for all healthcare personnel. The diagnosis is allowed rights in NAV's notification that was revised 01/06.

NAV suggest that the condition should be diagnosed using criteria formulated by the Centers for Disease Control and Prevention in USA. The CDC writes on its website that there is international concensus on the Fukuda definition, and it is used both for research and clinical use: http://www.cdc.gov/cfs/cme/wb1032/chapter1/overview.html

Internationally the Fukuda criteria have been criticised for being too broad and thereby including people with fatigue, but who do not have ME. The discontent with the Fukuda definition led to a strong need for clinical criteria. An international panel with experienced clinicians and researchers, with a mandate from Health Canada, therefore prepared clinical guidelines for diagnosis (Carruthers et al, 2003: http://www.mefmaction.net/documents/journal.pdf . These guidelines reflect the patients' situation best. President of the International Association of CFS/ME, Professor Dr Klimas PhD, has encouraged researchers and clinicians in using these, together with Fukuda criteria, in order to be able to compare research selection.

LOST VOICES

Lost Voices is a powerful addition to Invest in ME's library of educational aids. The book has been ongoing since our conference last May and Natalie, whose idea this was and who has devoted most of her waking hours to this project since our last London conference, has performed a quite amazing job. The book is of extremely high quality and is offered by Invest in ME at a reasonable price to allow more people to be able to purchase it.

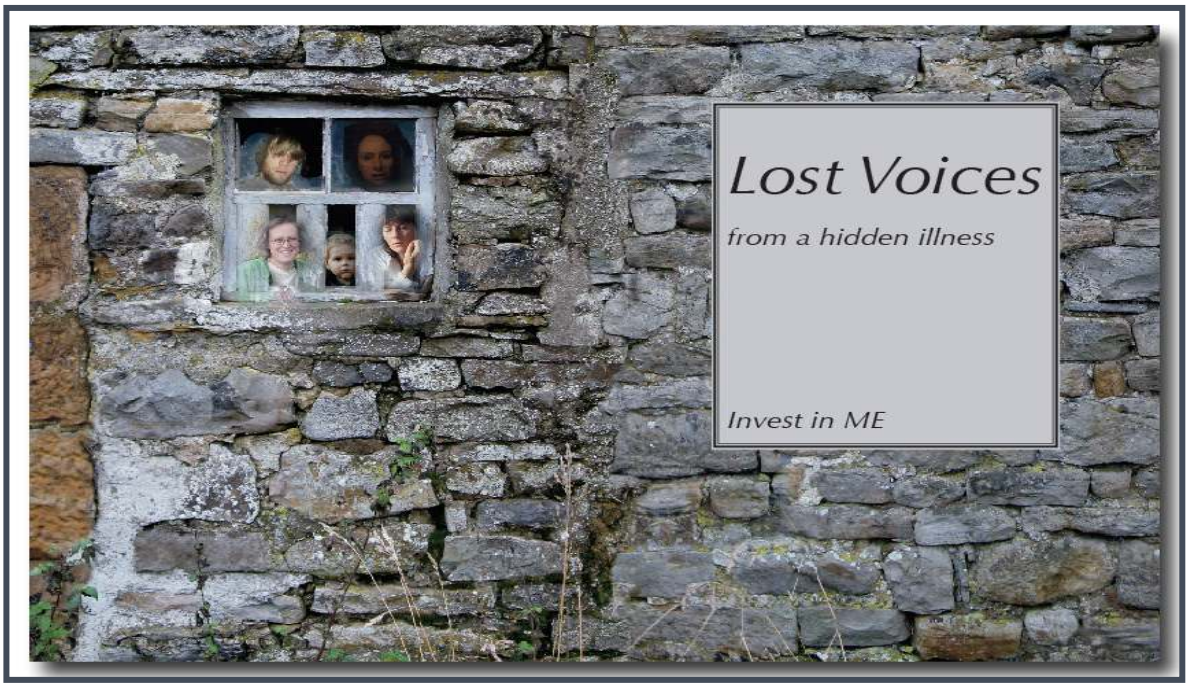
The early-purchase discount rate has been set at £8.00 (£9.00 for European delivery and £11.00 for delivery elsewhere) and includes postage and packaging. This applies to all payments received before 1st January 2009. After that date the price will be £10 (£11 for European delivery and £13 for delivery elsewhere).

The book is an A4 landscape size with a laminated card cover with pictures, mostly in colour.

With around 120 pages of stories, pictures and information this is without doubt the only book around which truly encapsulates the tragedy of this illness and the way in which people with ME are left to exist in a twilight zone - left to deal with this illness by themselves and with no hope of a future. The moving stories convey the real picture of ME.

And yet Lost Voices will show the resilient character of people with ME and their families.

The book also contains facts about ME with contributions from experts such as Dr. John Chia, Dr Leonard Jason, Dr Vance Spence and Annette Whittemore of the Whittemore-Peterson Institute.



If there is one book on ME that you buy then make it Lost Voices. Please buy this book - for yourself or for friends, relatives or your GP - or suggest it as a gift for others to buy.

This book will really make a difference.

More details can be found by clicking here [http://www.investinme.org/LostVoicesBook/IiME Lost Voices home.htm].

Support ME Awareness – Invest in ME